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ANNALS
OF
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Vol. XXV. MARCH, 1916. No. 1.

I.

THE DIAGNOSIS OF OTITIC CEREBELLAR ABSCESS.

By Alfred Braun, M. D.,

New York.

Cerebellar abscess is due, in the majority of cases, to suppurative disease of the middle ear. In fact, in a series of eighty-six cases of cerebellar abscess, collected by the writer from the literature since 1907 (which is the date of publication of Neumann's monograph on otitic cerebellar abscess), eighty-five were due to ear disease. The importance to the otologist of being able to diagnose this grave condition is, therefore, apparent. Unfortunately, it is a disease which often runs its course with practically no symptoms. Okada found that in ten per cent of cases of cerebellar abscess, death occurred before any symptoms of intracranial involvement appeared. In fourteen per cent of the cases the symptoms of cerebellar abscess were obscured by other otitic complications. In forty-two per cent the diagnosis was rendered very difficult on account of the presence of other intracranial complications, such as sinus phlebitis, pachy- or leptomeningitis, or temporosphenoidal lobe abscess. However, in the past few years important additions have been made to our knowledge of this subject, especially by Bárány.

A study of the anatomy and functions of the cerebellum will be of considerable assistance to us in diagnosing diseased conditions of this organ.

I will not go into the surface anatomy of the cerebellum, except to state that the lobes which are in relation to the posterior surface of the petrous pyramid are the biventral lobe and the posteroinferior lobe.

Bárány has been able to localize, in various parts of the cerebellar cortex, centers for movement of the various joints, in different directions. In the biventral lobe, just behind the internal auditory meatus, is the center for inward movement of the wrist joint. The center for position with palm downward is medial, and with palm upward, lateral. A little behind and above this is the center for inward movement of the shoulder joint, and just behind this for inward movement of the hip. Near the posterior pole of the hemisphere, at the medial end of the lobi semilunares superior and inferior, is the center for downward movement of the shoulder joint, and three to four centimeters outward from this point, on the edge of the hemisphere at the middle of the lobi semilunares superior and inferior, is the center for outward movement of the shoulder joint.

These facts were determined through observation of cases operated on for cerebellar abscess, through freezing of portions of the surface of the cerebellum, and through wounding of the cerebellum by operation. The study of cerebellar localization by animal experimentation has not been of very great value to us in this connection, for there is no close parallelism between the disturbances noted after destruction of portions of the cerebellum in the lower animals and those which the human clinic offers. In the human being the cerebellum has less influence on locomotion than in the lower animals. In the human being, when the motor centers of the cerebrum are destroyed, there is total paralysis. A dog can walk after total ablation of the cerebrum, provided the cerebellum is intact. In numerous cases of extensive disease of the cerebellum there are very few symptoms, probably because its functions are, in part, taken up by the cerebrum.

According to Bárány, the cerebellum contains centers in which the musculature of the body is represented according to the direction of movement. There are four centers: one for movement to the right, one for movement to the left, one for movement upward, and one for movement downward. Within these centers there is a subdivision according to joints, and according to positions of the joints. For example, in the center for movement to the right there are centers for the wrist, shoulder and hip joints, etc. And in the center for the wrist joint there is a center for movement with the hand in pronation, and one for movement with the hand in supination.

During rest there go from the direction centers in the cerebellum, to the muscles of the extremities, tonic impulses which hold the limbs quiet, the impulses from one center counteracting those from the other. When one center is destroyed the tonus from the opposite center overbalances, and there is spontaneous deviation or pointing-by of the limb. This deviation can only be elicited during voluntary motion—i. e., when the limb is held extended, as when an attempt is made to point to some object. The deviation is most marked after sudden destruction of a center. After a long time it may entirely disappear (compensation). The deviation is most marked when the eyes are closed, for if the patient is aware of his deviation he makes a voluntary effort to correct it.

If in an individual with a normal cerebellum a labyrinthine nystagmus is aroused by physiologic stimulation, there is a deviation of the extremities in the direction of the slow component of the nystagmus. This is known as the pointing reaction. But when there is a cerebellar lesion, the extremity affected by the lesion points correctly, in the presence of a vestibular nystagmus. In other words, there is a loss of the pointing reaction.

A lesion in one cerebellar hemisphere causes deviation only in the extremities of the same side. For example, with a lesion in the anterior portion of the right lobus biventer, there would be a loss of the inward tonus of the right arm. Consequently, on attempting to point at an object with the right arm, it would deviate outward. The left arm would point correctly. If, now, a horizontal nystagmus to the right is produced, either by cold water in the left ear, or by rotation to the left, the right arm would point correctly, whereas the left arm, which receives its stimuli from the intact cerebellar hemisphere, would deviate to the left.

That pressure upon the cerebellum has the same effect as destruction of portions of the cerebellum was well illustrated by a case presented by H. J. Davis before the otologic section of the Royal Society of Medicine in 1913. A patient had had a large abscess in the right cerebellar hemisphere, which was opened. There resulted a cerebellar hernia, which healed over. There was spontaneous rotatory nystagmus to the left. Pressure on the hernia increased the nystagmus to the left. Syringing the right ear with cold water increased the nystagmus to the left. Pressure on the hernia caused a still greater increase of this nystagmus. Syringing the left ear with cold water caused a nystagmus to the right. Pressure on the hernia arrested this nystagmus. There was spontaneous deviation of the right arm to the right. Pressure on the hernia increased this deviation to the right.

Bárány was able to verify this localization in twenty cases of cerebellar disease. In three cases of cerebellar cyst, observed by Horsley, there was loss of the pointing reaction to the left in the left arm, on arousing a horizontal nystagmus to the right. A diagnosis was made of a lesion in the left cerebellar hemisphere. Operation by Horsley showed cysts.

A case of cerebellar cyst, also of the left hemisphere, was diagnosed by Bárány. There was loss of the pointing reaction in the left arm to the right, and sometimes downward.

A case of bilateral cerebellar cyst was seen by Auerbach. There was loss of the pointing reaction in both arms. Operation. Cure.

A case of cerebellar tumor was seen by Tetens Haid. There was loss of the pointing reaction of the arm on the diseased side outward.

A case of cerebellar abscess was seen by Bárány. A boy was operated on for cholesteatoma and sinus thrombosis. An extradural abscess was found in the posterior fossa. Two days later there was deviation of the right arm outward, and loss of the pointing reaction inward. Cerebellar abscess was diagnosed. Operation. Cure.

A case of cerebellar abscess was operated by West. After operation, examination showed loss of the pointing reaction inward of the arm on the diseased side.

Four cases of cerebellar abscess were seen by Ruttin, Bárány, Claus and Brühl with the typical findings.

One case of cerebellar abscess was seen by Bruns with the typical findings.

Two cases of meningoencephalitis were seen by Ruttin and Bondy.

Two cases of operative injury of the cerebellum were seen by Bárány. In one of these, two incisions were made in the cerebellum: one at the posterior pole of the right hemisphere —i. e., at the medial end of the right lobus semilunaris superior and inferior—and the second, three to four centimeters outward from this point, on the edge of the hemisphere—i. e., in the middle of the lobus semilunaris superior and inferior. The effect of these operative lesions was a deviation of the right upper extremity upward and to the left, which indicated a lesion of the center for downward tonus of the right arm, and of the center for outward tonus of the right arm.

Two cases of incision of the cerebellum in front of the sinus, in suspected cases of cerebellar abscess, were seen by Bárány. There was deviation outward of the arm on the operated side.

A case of cerebellar abscess was seen by Holmgren. There was loss of the pointing reaction inward of the arm on the operated side.

In five cases the freezing experiment was done. That is, the cerebellar cortex was frozen by an ethylchlorid spray, in cases where there was a bone defect over the cerebellum, resulting from operation. Freezing the cerebellar cortex causes a temporary paralysis of the cerebellar centers.

Of these five cases, one was examined twelve times; two, twice; one, three times, and one, once. Seventeen times the frozen portion was the anterior and lateral aspect of the lobus biventer. In all these there was a deviation of the arm of that side. On freezing posteriorly and outward there was also deviation of the leg outward, and loss of the pointing reaction inward.

The uniformity with which the characteristic deviations and changes in the pointing reactions occur in so large a number of cases demonstrates, beyond any doubt, the value of this method of examination in cerebellar abscess.

Lesions of the cerebellar hemispheres cause deviations of the extremities. Lesions of the central lobe or vermis cause disturbances of the trunk muscles, with loss of equilibrium. When an artificial nystagmus is aroused, in the presence of a lesion in the vermis, the former will not influence the direction of the spontaneous falling; whereas, with an intact vermis, the direction of the nystagmus and the position of the head exert a definite influence upon the direction of the falling. For example, with a lesion of the vermis the patient falls when he attempts to stand up. The falling may be in any direction. We will assume that in the particular case under discussion the falling is backward. If, now, cold water be poured into the right ear, there will result a rotatory nystagmus to the left. This nystagmus will have no influence on the direction of the falling in the case under discussion, the direction remaining backward; whereas, if the vermis were intact, the falling, with the face forward, would be to the right.

The central gray matter of the cerebellum contains four pair of nuclei: the dentate nuclei, the nuclei tecti, the nuclei emboliformes, and the nuclei globosi. It is thought that the central nuclei have separate functions from the cortex of the cerebellum, just as the central nuclei of the cerebrum are considered as separate organs from the cerebral cortex.

The nucleus emboliformis and the nucleus globosus are merely prolongations upward of Bechterew's nucleus, which is one of the vestibular nuclei. In fact, all of the cerebellar nuclei are closely related to the vestibular nuclei.

The cerebellum is connected to the rest of the cerebrospinal system by means of three pairs of stalks or peduncles: a superior, a middle, and an inferior pair. The middle and inferior peduncles transmit, principally, the afferent fibers to the cerebellum, while the superior peduncles transmit the efferent fibers from the cerebellum. The afferent fibers to the cerebellum from the cerebrum reach the former through the middle cerebellar peduncles. The afferent fibers to the cerebellum from the spinal cord and medulla reach the former through the inferior peduncles or restiform bodies.

Two afferent tracts of fibers pass to the cerebellum from the spinal cord. They are Flechsig's tract and Gower's tract. Flechsig's tract is known as the direct cerebellar tract, and takes its origin from the cells of Clarke's column at the base of the posterior horn of the cord. The tract lies in the lateral column of the cord, at the posterior part of its margin. It passes up through the inferior cerebellar peduncle to the anterior portion of the cortex of the superior worm.

Gowers' tract, known as the anterolateral ascending cerebellar tract, takes its origin from the same cells, at the base of the posterior horn of the cord, as Flechsig's tract, and lies at the margin of the lateral column of the cord, just in front of Flechsig's tract. It passes through the pons, winds about the superior cerebellar peduncle at the point of emergence of the latter from the cerebellum, and terminates in the ventral portion of the vermis.

The inferior peduncle contains a nucleus known as v. Monakow's nucleus. It receives fibers from the posterior columns of the cord, and sends its fibers through the inferior peduncle to the same side of the cerebellum.

These afferent tracts to the cerebellar cortex transmit sensory impulses from the joints and muscles.

From the inferior or medullary olive, fibers pass through the arcuate bundles to the inferior peduncle of the opposite side, and through this to the cerebellar cortex and central nuclei.

Just internal to the inferior peduncle is a bundle of fibers known as the juxtarestiform body. It contains the cerebello-vestibular bundle, which consists of fibers that run between the vestibular nuclei and the cerebellar nuclei. The arcuate bundles also contain fibers that run from the vestibular nuclei to the cerebellum.

The afferent fibers to the cerebellum from the cerebrum pass through the middle peduncles. The fibers arise in the sensorimotor areas of the cerebral cortex, and pass down through the internal capsule and midbrain to the pons. Here they terminate about the pontine nuclei. From these nuclei fibers cross through the pons to the opposite middle peduncle, and then to the cortex of the cerebellar hemisphere.

Efferent fibers leave the cerebellum through two tracts. Fibers leave the dentate nucleus and pass through the superior peduncle to the red nucleus and the optic thalamus of the opposite side. From the thalamus, impulses reach the cerebral cortex through thalamocortical fibers. From the red nucleus, fibers pass down to the opposite side of the spinal cord, through

Monakow's bundle or the rubrospinal tract. This tract is situated in the lateral column of the cord in front of the pyramidal bundle. Its fibers terminate around the anterior horn cells of the cord. Thus each half of the cerebellum sends efferent fibers to the heterolateral cerebral cortex and the homolateral side of the cord.

The second efferent tract from the cerebellum also passes from the central nuclei, especially from the nuclei tecti. These fibers pass through the tractus uncinatus to the nuclei of Deiters and Bechterew, of the opposite side. From here fibers pass through the anterolateral segment of the cord to the anterior horn cells, and through the posterior longitudinal fasciculi to the oculomotor nuclei.

Thus we see that the cerebellum receives sensory impulses, sends out motor impulses, and is closely connected with the vestibular nuclei. Motor impulses reach the cerebellum from the cerebral cortex, and are there met by sensory impulses from the muscles and joints, and by impulses from the vestibular apparatus. These motor impulses are here acted upon in such a way by the centripetal impulses as to make them effective in maintaining the body equilibrium. Consequently, in cerebellar abscess there is no loss of muscular force; there are merely disturbances in the orderly execution of muscular movements.

Lesions of the fiber tracts in the cerebellum which connect the vestibular nuclei with the cerebellar nuclei, give rise to symptoms similar to those caused by lesions of the vestibular centers or of the vestibule itself.

Consequently, we may have, in cerebellar abscess, in addition to the general symptoms due to increased intracranial tension, and to the inflammatory process within the brain, symptoms of disturbed motility and symptoms of disturbances of the vestibular apparatus.

The disturbances of motility are the following:

1. Hypermetria.—The cerebellum seems to exercise an inhibitory influence on movements. In cerebellar lesions there is a suppression of this inhibition, resulting in unmeasured or immoderate movements. This is called hypermetria. It may occur in spontaneous movements, but is much more likely to occur in commanded movements. For instance, if a patient with cerebellar abscess is asked to touch the tip of his nose

with the end of his index finger, he will point past the nose. If a patient is seated, with the palm of his hand on the knee of the same side, and he is asked to supinate the hand, so that the back of his hand rests on the same spot where the palm was, the movement will be overdone, and the ulnar side of the hand will lie higher than the radial. A vertical line is drawn near the right edge of a sheet of paper. The patient is asked to draw horizontal lines, starting anywhere, but ending at the vertical line. The patient will continue all of his lines past the vertical line. In walking, at the beginning of the step, flexion of the thigh on the pelvis is more pronounced than normal, leading to excessive raising of the foot. At the end of the step the sole of the foot makes a greater noise than normal, in striking the floor, denoting excessive tension of the thigh.

The hypermetria is best brought out if the patient is asked to perform the movement quickly. For if it is done slowly he can correct the error. Vision has no effect on the movements. The same error is made with the eyes open as with the eyes closed. The direction of the movement is conserved. It passes the mark, and then deviates.

2. Cerebellar Asynergy.—If a patient with a cerebellar abscess is supported on either side, without his movements being influenced, and asked to walk, he will be stopped at his first step. The thigh is flexed and the foot is carried forward, but the upper part of the body does not move in harmony with the legs, the trunk remaining extended on the thighs. Thus, having placed his foot on the ground with much noise, at the end of the first step he can go no further. He is in danger of falling backward. It is necessary for the assistant to push the upper part of the body forward. Or the patient can help himself by pulling his trunk forward with his arms, his hands catching some support in front of him.

If a patient, standing up, is asked to bend his head back and curve his body backward, he falls, because he only bends the trunk back, without bending the legs on the feet, and the thighs on the legs. If a patient lies on his back, with his arms crossed on his breast, he cannot sit up. He flexes the thighs on his pelvis, and lifts the heels from the floor. These tests show that a patient with cerebellar abscess cannot synchronize the movements at the various joints in such a way as to perform an effective complex movement.

3. Adiadokokinesis.—This is the abolition or the diminution of the faculty of executing rapidly successive voluntary movements. In a cerebellar lesion, muscular force is intact. The patient can execute as rapidly as a normal individual the elementary movements, pronation or supination. But the complete movement (pronation and supination) he performs more slowly than a normal individual. The phenomenon becomes more manifest if the act is repeated a number of times.

The symptom is present only in an individual who can perform the isolated movements of pronation or supination with normal rapidity. For if he cannot do this, he cannot, of course, perform a rapid succession of the two movements. It is sometimes bilateral and sometimes unilateral. In the latter case it is on the same side as the cerebellar lesion.

4. Tremor.—On attempting to point to an object with one of the extremities, orientation is correct, but the movement is not uniform. There are oscillations in different directions. The tremor occurs only during voluntary contractions of the muscles. It varies in different individuals, and in the same individual at different times. It may diminish if the patient is left to himself and not observed, and if he executes movements slowly and carefully. On pointing to the nose with the finger, the movement to the nose may be straight, and then, at the end, there may be violent tremor.

5. Disturbances in Writing.—Ask the patient to make a dot on a sheet of paper with a pencil. He rarely succeeds at the first attempt. At first he goes through the paper, or breaks the point of the pencil. At the second attempt he stops above the paper. Finally, finding the surface of the paper, he makes, not a dot, but a line. Cerebellar patients place commas and not dots over their i's. This is hypermetria. If they are asked to draw a line of a certain length, they will extend it further. On attempting to draw a circle or an arc, as in the letter O or C, the figure is polygonal. In attempting to reproduce regular zigzags, as in M and N, the angles are badly formed and the lines irregular. Having arrived at the end of a line, the patient finds it difficult to begin a new line in the opposite direction. The irregularities in writing are the result of tremor, hypermetria, asynergy and adiadokokinesis.

6. Disturbances of Speech.—Speech is scanning, jerky, a little explosive, and sometimes dragging. It is similar to that in disseminate sclerosis.

7. Atony or Hypotony.—Hypotony is present in a few cases. It is sometimes bilateral and sometimes unilateral, in which case it is on the same side as the other symptoms. An experiment for determining the presence of hypotony of the extensors of the arm is as follows: ask the patient to flex his forearm, and oppose this by traction on his forearm. Suddenly stop the traction. In a normal person there results pronounced flexion, followed by a violent extension. In cerebellar lesions this extension is only indicated or altogether wanting.

8. Cerebellar Catalepsy.—This is a rare symptom. Voluntary muscles are held immobile for a long time, without being contracted, in certain positions where volitional equilibrium is realized. It often appears as follows: the patient lies on his back, thighs flexed on pelvis, legs lightly flexed on thighs, feet separated. When he assumes this attitude his legs and trunk oscillate in various directions, especially from left to right, and vice versa, but at the end of several seconds the body and lower limbs become fixed. This fixity is more marked than a normal individual can attain. It persists for several minutes, and causes no sensation of fatigue. In cerebellar affections, volitional kinetic equilibrium is diminished, while volitional static equilibrium is preserved or even increased.

9. Spontaneous Deviation of the Extremities, and Loss of Reaction Movements.—These symptoms, as worked out by Bárány, are of extreme importance in the diagnosis of cerebellar abscess. The method of examination is as follows: the patient is seated with his eyes closed. His arm is extended before him, and the index finger of his hand is made to touch the palm of the examiner's hand. The arm is then dropped until the hand touches the knee, and then raised until it again touches the examiner's hand. With a normal cerebellum the patient points correctly. With a lesion in the shoulder area of the cerebellar cortex, the arm will deviate either inward or outward. If the lesion is in the right biventral lobe, the right arm will deviate outward, or to the right. If the lesion is at the middle of the margin of the right cerebellar hemisphere —i. e., at the middle of the right posterosuperior and posteroinferior lobes—the right arm will deviate inward, or to the left. The former area contains the center for inward tonus of the shoulder joint, and the latter the center for outward tonus of

the shoulder joint. The left arm will point correctly. The deviation involves only the side of the body on which the lesion occurs.

The movements of the elbow joint are examined by resting the elbow on the back of a chair and moving the arm from the elbow. The movements of the wrist joint are examined by resting the wrist on the back of a chair and moving the hand at the wrist. The wrist joint is examined with the hand in pronation, and then with the hand in supination, there being a separate center for each position of the hand. The wrist center lies in front of the elbow center. The hip is examined in a similar way. The hip center is behind the arm center. These tests should be made repeatedly, in order to make sure that there really is a deviation, and the patient must not know when he has made an error in pointing, in order that he should not make a voluntary effort to correct the error.

Where the lesion in the cortex is a large one, many or all of the joints will be involved in the deviation. Where the lesion is small, only one or two joints will be involved.

Having determined the presence of a spontaneous deviation, the next thing is to test the reaction movements of the extremities. This is done by arousing an artificial nystagmus. If the patient is rotated to the left, he will have an afternystagmus to the right. If the patient's arms are now held in front of him, they will both deviate to the left—i. e., in the direction of the slow component of the nystagmus—provided the cerebellum is normal. If, however, the patient has a lesion in the right cerebellar cortex, involving the center for inward tonus of the shoulder joint, the right arm will point correctly, while the left arm will deviate to the left. Therefore, with destruction of this center there is a spontaneous deviation of the arm outward, and a loss of the reaction movement inward. Instead of rotation, the caloric test may be used to arouse the reaction movements, the head being tilted back sixty degrees in order to elicit a horizontal nystagmus.

If a cold caloric is done in the left ear, and the head held upright, the arms held in front of the body will deviate to the left. If the right arm is moved laterally, it will deviate upward. If the left arm is moved laterally, it will deviate downward. If the head is now rotated to the right shoulder, both arms held in front of the body will deviate downward. If the head is rotated to the left shoulder, both arms held in front of the body will deviate upward. The deviation depends upon the plane and direction of the nystagmus, and the position of the head. It is always in the plane of the nystagmus, and in the direction of the slow component of the latter.

Where there is a spontaneous deviation, but no loss of reaction movement, the lesion is probably remote, and causes impairment of function by pressure, and not by actual destruction of the center.

A deviation of the right arm to the right may be due either to destruction of the right center for inward tonus of the shoulder joint, or to hyperstimulation of the right center for outward tonus of the shoulder. In order to determine which of these conditions is present, the reaction movements of the right arm are compared with those of the left arm. If the reaction movement to the left of the right arm is wanting, there is destruction of the right center for inward tonus. If the reaction movement to the right of the right arm is greater than that in the left arm, there is a hyperexcitability of the right center for outward tonus of the shoulder.

When the deviation is in one direction only, and with increase in intracranial pressure it spreads to all the joints without taking a new direction, we are dealing with a cortical cerebellar lesion, rather than a lesion of the fiber tracts. This is analogous to monoplegia in the cortex of the cerebrum. When the deviation and loss of reaction movements is in more than one direction, we are probably dealing with a lesion in the central portion of the cerebellum, involving the fiber tracts.

10. Spontaneous Falling and Reaction Movements of the Trunk.—With an abscess involving the worm there is a tendency to fall. The falling may be in any direction. The direction of falling was noted in thirteen cases out of the eighty-six cases collected. Of these, ten cases fell to the side of the lesion, and three cases to the opposite side. If spontaneous nystagmus is present, the direction of the falling does not bear any definite relation to this nystagmus.

If an artificial rotatory nystagmus is aroused by means of cold or hot water in the ear, the patient falls in the direction of the slow component of the nystagmus, when the vermis is intact. For instance, if cold water is allowed to flow into the right ear, the patient falls to the right. If the head is turned to the right shoulder, the patient falls backward. However, if there is a lesion of the vermis, and the spontaneous falling is, for instance, forward, the direction of the falling would not be changed by the production of a caloric nystagmus. In other words, the falling reaction is wanting. There may be an absence of the falling reaction without spontaneous falling. The loss of the falling reaction may be in one or several directions. In the latter case the lesion would be larger and more centrally located than in the former case.

In order to differentiate between focal lesions and symptoms due to pressure from a distance, we must make a careful examination of the spontaneous symptoms as well as a careful functional examination. If there is spontaneous falling or deviation of the extremities, and on eliciting the proper nystagus, a falling or deviation in the opposite direction is elicited, we are dealing with a distant lesion. If, on examination at different times, there is sometimes deviation and falling and sometimes none, we are surely dealing with a distant lesion. If only a portion of the symptoms change, there is a combination of focal lesion and lesion at a distance. If, after a decompression operation, the symptoms disappear, there is a lesion at a distance. Constant symptoms, or symptoms which increase in severity, are due to focal lesions.

11. Hemiparesis.—This symptom occurs occasionally in cases of cerebellar abscess. It was noted in six cases out of the eighty-six collected from the literature. It is always on the same side as the cerebellar lesion.

12. Fixed Attitude of the Head.—In a few cases of cerebellar abscess the head is held stiff and rigid, and bent toward one shoulder. The cause of this attitude is not definitely known. It may be due to increased tonus of the neck muscles of one side. In experiments on dogs, where half of the cerebellum is removed, one of the most striking symptoms is the lateral deviation of the head and the lateral curvature of the entire body.

The symptoms which are due to destruction of the fiber tracts between the vestibular nuclei and the central nuclei of the cerebellum are nystagmus, vertigo and loss of equilibrium. The loss of equilibrium we have discussed above. The nystagmus due to cerebellar disease cannot be distinguished from that due to disease of the vestibular apparatus. It varies in its direction. Nystagmus was noted in forty-six out of the eighty-six cases. It was directed to the diseased side fifteen times, to the sound side seven times, to both sides twenty-two times, and in two cases the direction was not mentioned. A nystagmus to both sides, horizontal in one direction and rotatory in the other, is rather characteristic of cerebellar disease.

Although the appearance of cerebellar nystagmus does not differ from that of labyrinthine nystagmus, yet its behavior does. Labyrinthine nystagmus, when due to a diffuse labyrinthitis, gradually diminishes in intensity and stops in a few weeks. The direction of such a nystagmus is always away from the diseased ear. Cerebellar nystagmus remains the same or becomes worse. A nystagmus toward an ear of which the labyrinthine functions are destroyed cannot be labyrinthine in origin. It must be retrolabyrinthine, probably cerebellar. In some cases there is a dead labyrinth, with nystagmus toward the opposite side. During the course of the disease the nystagmus suddenly changes to the diseased side. This usually indicates a cerebellar abscess. The nystagmus of a circumscribed labyrinthitis is difficult to differentiate from that of a cerebellar abscess. In the latter case we often have other intracranial symptoms to guide us. Neumann recommends doing a labyrinthectomy in cases of doubt. If there was a circumscribed labyrinthitis the nystagmus will shift toward the sound side. If the nystagmus remains directed to the diseased side we are dealing with a cerebellar abscess.

If cold water be allowed to flow into an ear, and the flow stopped as soon as nystagmus sets in, the nystagmus will continue for one to two minutes, under normal conditions. In cerebellar disease the caloric nystagmus which is directed toward the affected hemisphere will continue for five or ten minutes, or even longer. This is called by Neumann, "enduring nystagmus," and is due to the loss of the inhibitory control which the cerebellum exercises over the vestibular centers.

Vertigo was present in fifty-six out of the eighty-six cases. There is nothing characteristic about cerebellar vertigo. Its intensity is not influenced by looking in different directions, as vestibular vertigo is.

A symptom due to disturbances of the sensory impulses was first described by Lotmar in the *Monatschrift für Psychologie und Neurologie,* 1908, and later confirmed by O. Maas and others. This is the underestimation of weights by the hand on the side of the cerebellar lesion. A series of weights is prepared, which are the same in size but different in weight. A weight is put into each hand, and the patient asked to judge whether they are equal or not. With a cerebellar abscess on the right side, a heavier weight in the right hand will seem to equal a lighter weight in the left hand. This symptom must be judged very carefully, for in right handed individuals the left hand normally overestimates weight slightly.

Changes in the reflexes were mentioned in seven cases out of the eighty-six. They were increased in five cases, and diminished in two cases.

Out of the above mentioned symptoms there may be only one or a few present, or there may be none at all. The cerebellar abscess may only be found at autopsy. Latent cases are of two kinds:

1. Where there never were symptoms. This might be due to separation of the nervous elements without their destruction. This is more probable in cases of tumor or hemorrhage than with abscess. It may be due to involvement of a silent area of the cerebellum.

2. Where there are symptoms at the beginning, which later disappear. This is due to compensation. Cerebellar symptoms may be made more marked and enduring by the association of lesions in compensatory organs (cerebrum, labyrinth and sensory paths). For instance, in disease of the cerebellopontine angle the eighth nerve is involved along with the cerebellum. This gives very severe symptoms.

The symptoms due to increased intracranial pressure are headache, vomiting, drowsiness, slow pulse, optic nerve changes, pupillary changes, and paralyses of the cranial nerves.

Headache was mentioned in seventy cases out of the eighty-six. It is the most constant of all the cerebellar symptoms. It is usually intermittent, as are most of the general cerebellar symptoms. There are often periods when the patient feels perfectly well. The location of the headache is not characteristic, but it is most often an occipital headache.

Vomiting occurred in forty-eight of the cases. It may occur with or without nausea. There are two types: projectile vomiting, due to increased intracranial pressure, and vomiting accompanied by nausea, which is due to involvement of the vestibulocerebellar tracts. Nausea without vomiting is mentioned in four cases.

Drowsiness was noted in thirty-two of the cases. Delirium or hyperexcitability is rare.

A slow pulse was present in thirty-eight cases. The pulse is always slow in relation to the temperature. When the temperature is normal or subnormal, the pulse may be as low as forty or fifty. When the temperature is raised by a complicating meningitis, the pulse is apt to be around seventy.

Optic nerve changes were present in sixteen cases. The condition found is usually an optic neuritis, but occasionally there is choked disc. It may be unilateral or bilateral, and, if unilateral, may be on the affected side or the opposite side. The impairment of vision is usually not great.

Inequality of the pupils was present in four cases, and in two cases the pupils were dilated and equal. Inequality of the pupils is usually due to the accompanying meningitis.

Paralyses of the third or sixth nerves were present eleven times. This is due either to basilar meningitis or to pressure on the nerves. In some cases there is present, instead of a third or sixth nerve paralysis of one side, a conjugate deviation of the eyes. This may be accompanied by a deviation of the head.

Facial paralysis or paresis occurred in twenty-seven cases. In the majority of these cases the paralysis was due to involvement of the facial nerve in the temporal bone, as the result of the accompanying labyrinthine suppuration. In a few cases it was due to pressure on the intracranial portion of the nerve.

Difficulty in swallowing occurs occasionally. This is due to pressure on the medulla.

The symptoms due to the inflammatory process in the cerebellum are temperature, changes in the cerebrospinal fluid, blood changes, and emaciation.

The temperature was noted in fifty-five of the cases. It was above 100° F. in twenty-three cases. It was between 98.6° F. and 100° F. in twenty-one cases. It was subnormal in eleven cases. The higher temperatures usually occur in the terminal stages when a meningitis has set in.

The cerebrospinal fluid was examined in twenty-four cases. It was normal in nine cases. It was normal, but under increased pressure, in two cases. It was cloudy and sterile in eight cases, of which three were under increased pressure. It contained bacteria in only five cases.

The blood changes of cerebellar abscess are not characteristic. There may be a leucocytosis with a high polynuclear count, or the blood picture may be normal. No cases of bacteremia have been reported in uncomplicated cases of cerebellar abscess.

Emaciation is always very marked in cerebellar abscess. The patients are cachectic in appearance. They are usually constipated, with coated tongue and fetid breath. There is rarely any bladder disturbance, except just before death.

The order of frequency of the symptoms noted in the eighty-six cases collected is as follows:

	Cases.		Cases.
Headache	70	Optic nerve changes	16
Vertigo	56	Incoordination of arms	12
Vomiting	48	Paralysis of third or sixth nerves	11
Nystagmus	46	Changes in the reflexes	7
Slow pulse	38	Hemiparesis	6
Drowsiness	32	Pupillary changes	6
Unsteady gait	32		
Facial paralysis or paresis	27		

CASE HISTORIES.

Case 1. Amberg, E.[1]—Female, aged four years. Otitis media suppurativa chronica. Admitted September 3, 1906.

September 6—Radical mastoid operation.

September 11—Incised lateral sinus. Free bleeding. Jugular ligation.

September 13—Temperature, 106.2° F.

September 15—Exposed dura of middle fossa. Dura looked healthy. Exposed cerebellum. Deep epidural abscess. Cerebellar abscess.

Patient discharged, October 10, 1906.

Case 2. Auerbach, J.[2]—Male, aged fifty-one years. Grippe. Pain in right ear. Right membrana tympani red and bulging. Slight tenderness over mastoid. Tenderness along anterior border of right sternomastoid. Pulse, 66; temperature, 99° F

Right ear, bad hearing. (Bárány noise apparatus used.) Membrana tympani incised. Drowsy. Mastoid operation two weeks later. Dura of middle fossa covered with granulations. Next day, slight rotatory nystagmus to right. Nausea, headache. Temperature, 102° F.; pulse, 90. Next day, chill; temperature, 103° F.; pulse, 108. Slight opisthotonos. Kernig marked. Temporosphenoidal lobe incised; no pus. Sinus found thrombosed. Abscess found in cerebellum. Jugular ligated. Exitus in a few hours.

Case 3. Bárány, R.[7]—Boy. Radical mastoid and sinus operation. Two days later, drowsiness, headache, and, on examination, the right arm pointed past, about fifteen centimeters to the right. Cold caloric in the left ear gave nystagmus to the right, and left arm and both feet deviated to the left, whereas, the right arm only deviated less to the right. Cold caloric in the right ear caused more marked deviation of the right arm to the right, and deviation of the other extremities to the right.

Two days later, opened cerebellar abscess, which lay just behind and external to the labyrinth. Two days later the spontaneous pointing-by had disappeared, but there was loss of pointing reaction to the left in the right arm. Six weeks later there was again normal pointing reaction to the left in the right arm.

Case 4. Barr, J. S.[8]—Infective labyrinthitis with septic thrombosis of lateral sinus. Opening in sinus wall leading to abscess cavity in cerebellum. Operation on labyrinth, cerebellum, sinus, and internal jugular vein. Death from purulent leptomeningitis. (Probably saccus empyema. A. B.)

Case 5. Barr, J. S.[8]—Subdural abscess, operated on. Later, cerebellar abscess was discovered and drained. Contained mixed staphylococci. Autogenous vaccins used. Recovery.

Case 6. Beck, J. C.[9]—Male, aged nineteen years. Otitis media suppurativa chronica in right ear since childhood. Six weeks ago, pain on right side of head, gradually increasing.

October 28, 1906—Pain, almost unbearable. Hearing markedly reduced. No dizziness. Temperature, 97° F.; pulse, 55 to 60.

November 1—Radical mastoid operation. Improvement of symptoms. Five days later, facial paresis. Pulse and temperature remained same. Nine days after operation patient suddenly died.

Postmortem—On opening tentorium a small opening was seen on surface of right half of cerebellum, close to median line. On enlarging this opening, escape of an ounce of pus. No disease in labyrinth. Meninges normal.

Case 7. Beck, K.[10]—Male, aged twenty-four years. Otitis media suppurativa chronica in both ears for ten years. For a week, vertigo and headache. Holds head directed toward left. Uncertainty in gait. Pulse, 50 to 60. Right ear has hearing. Positive caloric. Left ear, negative caloric. Lumbar puncture, turbid liquor, under pressure; sterile.

Radical mastoid operation, left. Sinus and dura of middle fossa bare. Puncture of sinus and dura of both fossæ negative. Two days later, exitus.

Postmortem—Thrombosis of sigmoid and inferior petrosal sinuses, right. Purulent circumscribed leptomeningitis about right cerebellar hemisphere. Cerebellar abscess, right. Perforation through inner wall of antrum into extradural space. Extradural abscess. Thrombosed vessels and pus cells in dura. Necrotic brain tissue between dura and abscess in cerebellum. Pial sheath infiltrated with round cells. Small abscess in pial sheath.

Case 8. Beck, K.[10]—Female, aged forty-six years. Grippe. Ten weeks later, earache, right. Four weeks ago, abscess behind right ear, incised. For past three days, vertigo and vomiting Head is held bent toward left side. Nystagmus on looking forward. Increased on looking to right. Stopped on looking to left. Both discs hazy. Right ear discharging. Has hearing. When eyes are closed, falls to the left. Caloric positive.

Mastoid operation. Epidural abscess in posterior fossa. Vomiting. Spontaneous nystagmus to sound side. Slight somnolence. Puncture of cerebellum medial to sinus; pus found; incision; drainage. Improvement for four weeks, then rise of temperature to 39.9° C. Sinus operation; thrombus. Three weeks later, symptoms in lungs; five days later, exitus.

Postmortem—Thrombosis of right transverse sinus, and a meningeal vein of right temporosphenoidal lobe. Gangrenous areas in both lungs. Empyema. Pneumonia. No pus in cerebellar abscess. Fibrous healing.

Case 9.—Biggs, G. M.[12]—Male, aged thirty-five years. Admitted September 17, 1908. Vertigo, two weeks. Tendency to fall to the right. Slight nystagmus in both directions. Otitis media suppurativa chronica, right, twenty years. Right grasp weaker than left. Some incoordination on right side in rapid rotation of hand. Dullness and apathy. Temperature, 98° F.; pulse, 60.

Radical mastoid operation. Cholesteatoma. External semicircular canal carious. At 10 p. m., unconscious. Temperature, 101.2° F.; pulse, 88.

Second operation. Trephine one and one-half inches behind and three-fourths inch below external auditory meatus. Cerebellum incised; pus. Patient improved. On September 22d, vomiting, drowsiness and irritability. Pulse, 60; temperature, 97°. Left pupil more dilated than right. Respirations, 16. Lumbar puncture showed normal fluid. Temporosphenoidal lobe and cerebellum explored with negative results. Exitus, September 25th.

Postmortem—Ventricles distended with clear fluid. Cerebellar abscess.

Case 10. Black, W. D.[13]—Male, aged fourteen years. Otitis media suppurativa acuta, right, four weeks. Headache above right ear, radiating to frontal region. Vomiting; vertigo. Pulse, 60 to 70; temperature, normal. Drowsiness. Paracentesis. Five days later, Schwartze-Stacke operation. Pus in antrum. Sinus exposed. Looked normal. Temporosphenoidal lobe exposed and explored. No pus found. Fifteen days later, brain prolapse resected. Opening filled in with silver screen. Death on table.

Postmortem—Abscess in right lobe of cerebellum.

Case 11. Bourguet.[14]—Female, aged fifty-four years. Otitis media suppurativa chronica, right, since infancy.

September 10, 1912—Headache, vertigo. Walking difficult.

September 20—Curettage of middle ear. Headache became more violent. Vomiting.

September 30—Curettage again.

December 3—Headaches worse. Spontaneous nystagmus to both sides, more marked to right. Caloric negative. Right eighth nerve paralyzed. Soft palate moves less on right than on left side. Paralysis of hypoglossal, right. Tongue deviates to right. Pulse, 100; temperature, normal. Falls to right. Adiadokokinesis.

Operation; cholesteatoma. Descending portion of facial is exposed. Sinus exposed. Labyrinthectomy. Cerebellum incised; pus. Cure.

Case 12. Braun, A.[15]—Male, aged fifty-eight years. Admitted July 3, 1909. Otitis media suppurativa acuta, right, for two weeks. Mastoid tenderness. Temperature, 102° F. Simple mastoid operation. Perisinus abscess. Hole in outer wall of sinus. Clot in sinus. Jugular resection. Clot evacuated from sinus. Temperature normal next day. Remained normal for thirteen days.

July 16—Headache. Temperature and pulse normal.

July 19—Headache. Temperature, 102.4° F.; pulse, 72.

July 23—Chill; apathy. In afternoon, all the signs of a well marked meningitis. Lumbar puncture showed a turbid fluid containing pus cells and pneumobacillus of Friedländer.

July 24—Exitus.

Postmortem—Pia bathed in thick creamy pus, extending to base of brain and down into spinal canal. Ventricles full of pus. Superficial abscess in right lobe of cerebellum, a short distance behind obliterated sinus.

Case 13. Brühl, H.[16]—Female, aged twenty-seven years. Cholesteatoma, facial paralysis, ataxia, deafness, dead labyrinth on diseased side, marked nystagmus to diseased side, and increased vestibular irritability of the opposite side. Spontaneous deviation of the arm of diseased side outward. The inward reaction after turning was wanting. In the opposite arm the reaction was present. Abscess could not be found at operation.

Postmortem—Cerebellar abscess three centimeters from the surface, in the lobus biventer.

Case 14. Curtillet and Aboulker, H.[18]—Male, aged forty-seven years. Otitis media suppurativa chronica, right, for two years; bloody discharge. Deafness for a long time. Three months before, at the beginning of 1910, intense vertigo.

In November, 1910, intense earache.

January 6, 1911—Vomiting and headache. Cannot stand. Three weeks before, facial paralysis, right. Right leg weaker than left. No adiadokokinesis. No dysmetria. Kernig. Spontaneous nystagmus in both directions. Pulse, 60; temperature, 36.2°. Lumbar puncture shows cerebrospinal fluid under pressure and clear; sterile; contains a few white cells. Positive caloric. No hearing, right and left.

January 11—Operation. Puncture of temporosphenoidal lobe, negative.

January 15—Second operation. Incision of cerebellum; pus. Cure.

Case 15. Davis, H. J.[19]—Boy, aged eight years. May 20, 1912, simple mastoid operation, right. Large epidural abscess in posterior fossa.

June 10, three weeks later, drowsiness, optic neuritis and nystagmus to the opposite side. Evacuated large abscess in right cerebellar hemisphere. Hemorrhage from lateral sinus. Hernia of cerebellum. It receded after a time until it was flush with surface of head and skinned over. Cure.

Spontaneous rotatory nystagmus to left. Pressure on hernia increases nystagmus to left. Syringing diseased ear with cold water causes nystagmus to the left. While syringing, pressure on the hernia increases intensity of the nystagmus to the left. Syringing sound ear with cold water for one minute causes horizontal nystagmus to the right for two minutes. On another day, horizontal nystagmus to right is arrested by pressure on the hernia.

Pointing Tests.—Up and down test, deviation to right. Side to side test, accurate. Pressure or application of ice to the hernia increases the deviation to the right, in the up and down test. Does not alter the accuracy of the side to side test. In pointing tests with feet there is also a deviation to right in up and down test. No change in side to side test.

Case 16. Dench, E. B.[20]—Male, aged four years. No history obtainable. Had mastoiditis, right. Simple mastoid operation. No dura exposed. Death, four days later.

Postmortem—Small perforation in inner table, over lateral sinus. Perforation led through sinus, which was thrombosed, into abscess cavity, in cerebellum. The patient presented no symptoms of cerebellar disease.

Case 17. Dench, E. B.[20]—Male, aged twenty-six years. Double otitis media suppurativa chronica for eight years. In the spring of 1905, emaciation. Suppuration on left side more profuse. In October, 1905, radical mastoid, left. Facial paralysis three days later, which almost entirely disappeared a month later. Five months later, nausea and vomiting, diplopia and recurrence of the facial paralysis. Marked incoordination of upper and lower extremities. Paresis of left external rectus and left superior oblique. Slight congestion of retinal veins of both eyes. Temperature, subnormal; pulse, 60 to 70. Polynuclears, 72 per cent. Headache and slight rigidity of neck.

Operation. Cerebellum exposed and explored behind sinus. No pus found. Death three days later.

Postmortem by Dr. G. S. Dixon.[24]—Dura normal. Seropurulent leptomeningitis, more intense on left side. The arachnoid was studded with very fine points, suggesting tuberculosis. No meningitis at base. Both lateral ventricles contained much bloody serum. Small necrotic area on upper surface of left quadrate lobe of cerebellum. On cutting through this an abscess was found, involving both hemispheres and the worm. Nodular tumor involved seventh and eighth nerves, in the internal auditory canal. Fibrosarcoma. No erosion of internal auditory canal. Cochlea completely destroyed by growth, also vestibule.

Case 18. Dench, E. B.[20]—Male, aged thirteen years. Acute exacerbation of otitis media suppurativa chronica, right, with mastoid symptoms. Radical mastoid operation. Evacuation of perisinus abscess. Two days later, slight temperature elevation. Leucocytosis, 33,600. Polynuclears, 84 per cent. Temperature rose to 104° F., and he became stupid and complained of severe headache. Cerebellum was exposed both in front of and behind sinus, and a cerebellar abscess was found. Through and through drainage. Patient died twenty-four hours later.

Case 19. De Stella.[22]—Female, aged twenty-three years. Otitis media suppurativa chronica, right. Vertigo and disturbances of equilibrium. No nystagmus. Radical mastoid caused no improvement. Vomiting, occipital headache, slow pulse, drowsiness. Papilledema at end, and crossed hemiplegia (right facial and left extremities). Patient had hearing in right ear, and static labyrinth responded to stimulation. A cerebellar abscess was diagnosed, but operation was refused. Autopsy disclosed an abscess in the worm, and in a large portion of the right cerebellar hemisphere, breaking through the middle peduncle into the pons.

Case 20. Dighton, Adair.[23]—Female, aged thirty years. Admitted May 7, 1913, with subacute mastoiditis, left. Temperature, 98° F.; pulse, 100. Discharge for six months. Labyrinth normal. Heath operation. Next day temperature 103° F.;

pulse, 92. A week later, slight nystagmus toward diseased side. Left hand pointed by to the left. Right hand pointed correctly.

Second operation. Incised left cerebellar hemisphere. Pus, which contained pneumococci and diphtheroid bacilli. Recovery.

Case 21. Dortu, P.[25]—Male, aged forty-one years. On March 11th, sudden earache, right. Paracentesis, repeated in a few days. Repeated several times during two months.

May 17—Pain over mastoid, in neck, vertigo and nausea.

May 21—Operation. Perisinus abscess. The following day patient felt well. Three days later, temperature, 36.5° C.; pulse, 60 to 65.

May 25—Vomiting, nausea. Hearing good. Eye grounds normal. No disturbances of equilibrium.

May 29—Restless and stuporous. Legs flexed. Lies on side of lesion. Headache. Facial paresis, right. Slight nystagmus to right on looking to right. Reflexes exaggerated, especially on right. Right arm shows incoordination. Left arm normal. Walking is impossible. Falls to side of lesion. Cerebrospinal fluid contains a few lymphocytes and polynuclears. Caloric negative on affected side.

Second operation. Incision of cerebellum; pus. Improvement. Healed without incident.

Case 22. Dortu, P.[26]—Male, aged eleven years. Admitted May 25, 1908. A week ago, chill followed by sweat, and pain in left ear and discharge. Four days later, another chill, and pain extended to half the head. Vertigo, nausea and vomiting. Head is bent to left shoulder. Emaciation. Large granulation in left canal. Left facial paralysis. Deviates to side of lesion. Pulse, 100; temperature, 36.7° C. No hearing in left ear. Lumbar puncture shows normal fluid.

May 26—Operation.

May 28—Pulse, 54; temperature, 36.4° C. Rigidity of neck. Paresis of left arm and leg. Increased knee jerk.

May 29—Second operation. Cerebellum incised. Negative. Next day, exitus.

Postmortem—Posterior surface of petrosa, behind internal auditory meatus, shows greenish discoloration. A little pus under dura at this point. Lateral ventricles dilated. Fluid clear. Abscess in left lobe of cerebellum. Fistula in external semicircular canal. Pus in entire labyrinth.

Case 23. Dougherty, D. S.[27]—Female, aged nineteen years. Seen November 8, 1908. Otitis media suppurativa chronica, left, for ten years. A week before, earache, vertigo, chill, and nausea. Temperature, 104° F.; pulse, 140. Mastoid tenderness. Slight nystagmus.

Mastoid operation. Perforation in bony wall over sinus. Sinus wall gangrenous and perforated. Contained clot. Clot removed.

November 15—Restlessness, chills and headache. Temperature, 101.8° F.; pulse, 76. Jugular resection.

November 21—Temporosphenoidal lobe incised; negative. Cerebellum incised; pus.

November 28—Collapse. A portion of occipital bone removed, and through and through drainage instituted. Recovery.

Case 24. Dougherty, D. S.[27]—Male, aged fifty years. Otitis media suppurativa chronica, right. For two weeks, earache. For one week, facial paresis. Admitted July 27th.

August 5—Chill. Temperature, 103.4° F. Vertigo and nausea.

August 6—Radical mastoid. Sinus incised. Clot evacuated.

September 7—Chill and nausea. Vertigo. Nystagmus to left. Neuroretinitis. Spinal fluid normal. Temporosphenoidal lobe incised; negative. Cerebellum incised; pus. Recovery.

Case 25. Forgue, E., and Rauzier, G.[28]—Male, aged twenty-four years. Admitted October 21, 1911. Otitis media suppurativa chronica, left. Pain in left ear for ten days, and headache and vertigo. On admission, temperature, 39.2° C.; pulse, 68. Mastoid tenderness. Vomiting.

October 26—Lumbar puncture. Cloudy fluid. Numerous leucocytes. Sterile.

October 31—Exitus.

Postmortem—Cerebellar abscess in left hemisphere. On posterior surface of petrosa, dura is thickened. Mastoid full of pus. Fistula leading to posterior surface of petrosa.

Case 26. Glegg, W.[29]—Male, aged thirty-three years. Otitis media suppurativa chronica, right. Polyp removed. Admitted end of November, 1911. Pain in right ear for a week, with vomiting. Temperature, 100°; pulse, 108. Vestibular reaction present on both sides, but less readily induced on right side. Right optic neuritis. Paresis of right sixth nerve. Next day, radical mastoid. Sinus injured. Four days later, jugular ligation. Sinus opened and white thrombus removed. Deep extradural abscess in posterior fossa opened. Knee jerks slightly increased. Spontaneous nystagmus on looking to right.

December 22—Cerebellum and temporosphenoidal lobes explored. No pus found. Exitus.

Postmortem—Abscess in middle lobe of cerebellum. A second abscess in right temporosphenoidal lobe, immediately adjacent to the cerebellar abscess. The tentorium between was intact.

Case 27. Hibler, E.[32]—Male, aged forty-nine years. Admitted January 23, 1909. Otitis media suppurativa acuta (?), right, for ten weeks. For five weeks, pain. Two days, vertigo and tendency to fall to the right. For ten days, facial paralysis, right. Marked horizontal nystagmus to the right. Slight nystagmus to left. Granuloma in right canal. Somnolence. Pulse, 60. Death, January 24th.

Postmortem—Abscess in right cerebellar hemisphere. Purulent lepto- and pachymeningitis. Osteitis and periostitis of right temporal bone, combined with carcinomatosis. Thrombosis of right sigmoid sinus.

Case 28. Isemer, F.[33]—Male, aged thirty-nine years. Admitted March 15, 1907. Otitis media suppurativa chronica, right. Two weeks ago, earache, right, and vomiting. Day before admission, vertigo and slight facial paresis, right. Nausea. Right side of occiput and back of neck sensitive. Eye grounds normal. Marked nystagmus, especially on looking to right. Knee jerk lost on right side, slight on left. Standing with eyes closed, sways to right and backward. Heard loud voice, right; Weber to left. Temperature, 37.5° C.; pulse, 82.

March 15—Radical mastoid. Cholesteatoma. Sinus covered with granulations. Fistula in external semicircular canal.

March 17—Diplopia. Paresis of internal rectus and obliquus.

March 18—Increased headache.

March 19—Sensorium cloudy. Pulse, 64. Cerebellum incised; pus.

March 22—Chills. Temperature, 39.2° C. Jugular ligation. Sinus incised; clot.

March 25—Optic neuritis.

April 1—Erysipelas starting from nose.

April 3—Deviation conjugee.

April 11—Exitus.

Postmortem—Large abscess in right cerebellar hemisphere, surrounded by encephalitis. Broken down thrombus in right lateral sinus. Pus in jugular bulb. Fistula in promontory. Stapes is present. On removing it, pus flows out of oval window. Facial exposed. No pus in internal auditory meatus. Small abscess in region of saccus. Outlet of aqueductus vestibuli is enlarged and blackish.

Microscopic examination shows cochlea and vestibule entirely destroyed. Aqueductus vestibuli is dilated (more than one millimeter) and full of pus. Saccus cannot be found.

Case 29. Jacques, P., and Goulet, G.[34]—Male, aged forty years. Admitted February 17, 1910, with grippal otitis, right, for three weeks. Temperature, 41.2° C. Rapid pulse. Vertigo, chills, headache. Semisomnolence. Facial paresis, right, and spontaneous horizontal nystagmus. Right ear contains polypi. Slight mastoid tenderness. Complete deafness, right.

Radical mastoid operation. Cholesteatoma. Small fistula in ampulla of external semicircular canal. Small fistula in facial canal, behind and above oval window. Lumbar puncture showed clear fluid.

February 19—Chill. Temperature, 40° C. Nystagmus reappeared.

February 20—Exposed portion of sinus wall is gangrenous. Incised. Pus and clot.

February 21—Vertigo.

February 22—Headache. Temperature, 40° C. Cerebellum incised; no pus.

February 23—Exitus.

Postmortem—Abscess in inferior portion of right cerebellar hemisphere.

Case 30. Karsner, H. T.[35]—Male, aged sixty years. November 1, 1910, noticed that in walking his body bent to right, and he had tendency to turn to right. Pain in occiput and back of neck. Coordination in movements of hand was poor. Knee jerks exaggerated. Ankle clonus. Grip weaker in right than in left hand. Slight slowing of pulse. No temperature. Death, November 16th.

Postmortem—Gangrene at base of right lung. Middle lobe of cerebellum contained large abscess. Pus showed pure culture of staphylococcus cereus albus. Lung showed same organism.

Case 31. Kelson, W. H.[36]—Female, aged forty-three years. Operated February 2, 1910, for nasal polypi.

February 7—Otitis media suppurativa acuta, left.

March 1—Temperature, 100.5° F.; pulse, 78. Wasted. Drowsy. Vertigo. Left pupil larger than right. Both reacted. Coarse slow nystagmus to left seen on one occasion. Pus and granulations in left ear. Has hearing. Knee jerk more marked on left. Vomiting, headache. Prefers to lie in flexed position, on right side. Constipation. Positive caloric, left. Limbs of left side weaker than right.

March 3—Cerebellum explored. Abscess found in left hemisphere. Contained streptococci.

March 28—Exitus.

Postmortem—Showed abscess surrounded by edema, and a small abscess on dorsal surface of left cerebellar hemisphere.

Case 32. Kerrison, P. D.[37]—Female, aged sixteen years. Otitis media suppurativa chronica, both ears. First seen February 9, 1910. Acute mastoiditis, right. Temperature, 100.4° F. Dizzy for three or four days.

Operation, February 9. Perisinus abscess.

February 14—Headache and vertigo. Slight rotatory nystagmus in lateral positions.

February 20—Negative caloric. Good hearing.

February 23—Frontal headache. Vertigo, vomiting. Nystagmus to right more marked. Falls to right. Falling not influenced by position of head. Hypermetria, right. Speech thick. Adiadokokinesis, right. Left wrist rotates four times while right wrist rotates once.

March 3—Eye grounds normal.

March 6—Second operation. Cerebellum behind sinus; pus.

May 10—Discharged cured.

Case 33. Kirmisson, M. E.[38]—Male, aged eleven and one-half years. Admitted January 13, 1902. Otitis media suppurativa chronica, right. Fistula over mastoid, right, following an abscess incised two weeks before. Purulent discharge from ear. Prostration, headache, constipation. Pulse, 98; temperature, 37.2° C. Vomiting.

January 15—Mastoid operation. Hyperesthesia. Weakness of all four limbs. Vertigo. Objects turn from his left to his right. Knee jerks lost. Kernig.

January 20—Temporosphenoidal lobe incised; nothing found. Cerebellum incised; pus.

March 24—Cure. Pus from abscess showed colon bacillus.

Case 34. Knapp, A.[39]—Male, aged twelve years. Otitis media suppurativa chronica, right, two and one-half weeks before admission; subperiosteal abscess was opened. Seen March 27, 1906. Drowsy, pupils dilated, optic neuritis. No hearing, right. Temperature, normal; pulse, 55. Rigidity of neck. Lumbar puncture showed cloudy fluid. Mastoid operation; cholesteatoma. Temporosphenoidal lobe incised; no pus. Deep epidural abscess in posterior fossa. Opening in dura. Opening enlarged. Large abscess in cerebellum. Respiration very bad during operation. On evacuating abscess, breathing improved. Exitus a week later.

Postmortem—Purulent exudate over base of brain, covering optic chiasm, and extending back into spinal cord. Abscess cavity in anterior part of right cerebellar hemisphere. Lateral ventricles dilated, and contain turbid fluid. External semicircular canal destroyed by cholesteatoma. Streptococci in spinal fluid.

Case 35. Labouré, J.[41]—Male, aged twenty-three years. First seen July 13, 1910. Fifteen days before, headache. Vomited two or three times. Vertigo. In left ear, cicatrices in membrana tympani, and small perforation with a little mucopus. Temperature, 38° C. No mastoid tenderness. Negative Rinné, left. Weber uncertain. Simple mastoid operation. Cells contain pus. Posteroinferior wall of antrum is sequestrated.

Patient lies on left side, holding his head in his hands. Mentality clear. Memory good. Answers questions slowly. Spontaneous nystagmus to diseased side. With noise apparatus in right ear there is some hearing in left ear. Rotation to right gave only two nystagmic movements to left. When he walks he drags his left leg and deviates to left; more so the longer he walks. When he is still he spreads his legs apart, enlarging his base of support. He can stand with his eyes closed, but loses his equilibrium during rotation. Jumping forward or backward is absolutely impossible. Muscular power diminished on left side. Diagnosis of cerebellar lesion is made, with or without a labyrinthitis.

July 25—Second operation. Epidural abscess found over cerebellum. Large hole in dura leading into cerebellum. Cortical cerebellar abscess. After operation, nystagmus persisted.

Left pupil dilated. Ptosis, left. Divergent strabismus. Third nerve paresis, left. Patellar reflex exaggerated. Slight rigidity of neck and slight Kernig. No Babinski. Lumbar puncture showed clear fluid. The pus from the cerebellar abscess contains only scattered Gram positive intracellular cocci. Cerebrospinal fluid showed a few white and red cells; no organisms. The ocular paralysis and nystagmus gradually disappeared. Patient discharged in a month.

Case 36. Lannois,. M.[42]—Male, aged thirty-nine years. Otitis media suppurativa chronica, right, since seven years of age. One month ago vertigo, which caused him to fall, without losing consciousness. Vomited two days. For past twelve days facial paresis, right. Spontaneous nystagmus to both sides, most marked to left. Not modified by caloric in right ear. Temperature around 38° C. Labyrinthine operation had no effect on symptoms. Two weeks later, paresis of right abducens. Trephining and puncture over cerebrum and cerebellum; no pus found. Autopsy showed a double abscess in cerebellum, which was not reached, on account of its situation high up and in front.

Case 37. Laurens, P.[43]—Male, aged twenty-one years. Admitted January 18, 1909. Otitis media suppurativa chronica, left, since infancy. For past week, violent headache, left. Vertigo, vomiting. Temperature, 39.7° C.; pulse, 72. Knee jerks diminished. Mastoid tenderness. Diminished Schwabach. Weber to right. Slight spontaneous nystagmus to sound side. Caloric positive. Cerebrospinal fluid was under normal pressure; a trifle cloudy; contained lymphocytes and polynuclears, but no bacteria.

January 21—Radical mastoid.

January 27—Epileptoid attack.

January 28—Temperature, 36° C.; pulse, 60. Torpor. Nystagmus to diseased side, in addition to nystagmus to sound side. Exitus.

Postmortem—Meninges normal, except on posterior surface of petrosa, where there are adhesions between dura and pia. Dilatation of lateral ventricles. Abscess in left lobe of cerebellum. The portion of the petrosa between labyrinth and sinus, corresponding to inner antral wall, is diseased.

Case 38. Laurens, P.[43]—Female, aged nineteen years. Admitted in comatose condition. Rigidity of neck. Kernig. Cutaneous hyperesthesia. Photophobia. Inequality of pupils. Exaggerated knee jerks. No nystagmus. Temperature, 38° C.; pulse, 54. Otitis media suppurativa chronica, right. Cerebrospinal fluid cloudy; contains polynuclears and anaerobic bacteria. Exitus twelve hours later.

Postmortem—Meninges normal, except on posterior surface of petrosa. In right lobe of cerebellum an abscess. Spinal canal full of pus. Disease in inner wall of antrum between labyrinth and sinus.

Case 39. McBride, P.[44]—Male, aged twenty-three years. Seen October 14, 1907, Otitis media suppurativa chronica, right. Four days before, pain. Edema over mastoid. Temperature, normal; pulse, 84. Right ear deaf.

Seen again January 14, 1908. Vertigo for two weeks. Vomiting. Nystagmus on looking to the right.

January 16—Radical mastoid.

February 5—Incised lateral sinus; clot. Drowsiness.

February 17—Nystagmus on looking to the left. Exploration of cerebellum and temporosphenoidal lobe; no pus. Symptoms improved.

March 1—Again explored; no pus.

March 7—Abscess found in cerebellum. Recovery.

Case 40. Mathewson, G. H.[46]—Male, aged nineteen years. Seen March 14, 1912. Otitis media suppurativa chronica, left. On February 23d, headache and vomiting. Apathy. On looking to right, marked nystagmus. Vertigo. Pupils dilated.

March 16—Operation; cholesteatoma. Inner table gone over sinus and cerebellum. Hole in cerebellar dura, from which pus was oozing. Opening enlarged and large cerebellar abscess evacuated. Cure.

Case 41. Michaelsen, U.[47]—Female, aged forty-three years. Polyp removed from right ear. Next day, headache, drowsiness, vertigo, nausea. Right ear, no bone conduction. Negative Rinné. Watch is not heard. Positive caloric.

November 26, 1909—Radical mastoid. No stapes. Probe passes easily into vestibule. On probing downward and backward the eyes move downward. On probing backward the eyes deviate to the diseased side. On probing upward the eyes deviate upward. On probing forward there is no movement of eyes. Labyrinth operation.

November 26—Vomiting. Marked escape of cerebrospinal fluid. Rotatory nystagmus to left. Temperature, 37.2° C.

November 27—Apathy. Pulse slow. Slight rigidity of neck. Lumbar puncture shows turbid fluid. Resection of remainder of posterior surface of petrosa; no pus. Internal auditory meatus opened.

November 29—Vomiting. No nystagmus. Pulse slow.

December 4—Right facial paralysis.

December 11—Cerebellum aspirated; pus found. Incision. Evacuation of one and one-half ounces of pus.

January 4, 1910—Patient discharged cured. Completely deaf, right.

Case 42. Michaelsen, U.[47]—Male, aged forty-three years. Otitis media suppurativa chronica, right. Has bone conduction. Weber to right. Rinné negative. Watch heard at seven centimeters. Marked nystagmus to sound side. On irrigating with cold water, violent dizziness.

December 9, 1909—Radical mastoid operation; cholesteatoma in antrum. Small fistula in ampullated end of external semicircular canal. On introducing probe, movement of eyeballs.

January 29, 1910—Readmitted with headache, vertigo and vomiting. Tinnitus. Nystagmus to left. Has bone conduction. Watch is heard at nine centimeters.

February 1, 1910—Labyrinth operation; cerebellar abscess found. Lumbar puncture shows turbid fluid. Exitus.

Postmortem—Purulent leptomeningitis. Walnut-sized abscess in right cerebellar hemisphere. Recent thrombus in right lateral sinus. Puncture into cerebellar abscess entered lateral ventricle, which was dilated. There was a circumscribed labyrinthitis, which caused infection through ductus endolymphaticus to saccus. This led to cerebellar abscess. Cochlea was not involved.

Case 43. Michaelsen, U.[47]—Male, aged fifteen years. Right, tenderness over mastoid. Canal full of pus and granulations. No bone conduction. Rinné negative. Optic nerves somewhat blurred.

June 25, 1910—Radical mastoid operation; cholesteatoma. Fistula in ampullated end of external semicircular canal. Perisinus abscess. Fistula in wall of sinus. Jugular ligation. Lateral wall of sinus excised. On passing probe into fistula in external semicircular canal, no movements of eyes. Exposure of cerebellar dura beyond saccus. Vestibule opened. Internal auditory canal opened.

June 26—Vomiting. Pulse, 50. No temperature. Somnolence.

June 30—Puncture of cerebellum; pus. Incision and drainage of a superficial abscess.

July 8—Headache. Lumbar puncture shows clear fluid. Puncture of cerebellum discloses no pus. Choked discs increased.

July 16—Exitus.

Postmortem—Purulent leptomeningitis. Encephalitis, especially of cerebellum, probably starting from saccus.

Case 44. Michaelsen, U.[47]—Male, aged thirty-two years. Otitis media suppurativa chronica, both. Two weeks ago, headache and vertigo. Had polyp removed from ear. Headache worse. Left facial paresis and mydriasis. Reflexes increased. On walking, deviation to the right. With Romberg, falls to right and backward. Bilateral radical mastoid; labyrinth operation, left. Large fistula in ampullated end of external semicircular canal. On probing, no movement of eyes. Exudate in vestibule. Dura of posterior fossa normal. No escape of liquor on opening internal auditory meatus. Facial exposed in horizontal portion. Lumbar puncture shows clear fluid; no pressure. Vomiting. Headache improved. Somnolence. Incision of cerebellum; pus; drainage. Three days later, marked optic neuritis, right. Left, slight changes in disc. Puncture of right temporosphenoidal lobe. At depth of three and one-half centimeters, turbid liquor, which evidently comes from lateral ventricle. Incision. Glass drain. Enormous flow of liquor. Next day, exitus.

Postmortem—Suppuration in left labyrinth, extending to dura and pia of posterior fossa. Abscess in left cerebellar hemisphere. Internal hydrocephalus.

Case 45. Michaelsen, U.[47]—Female, aged thirty-four years. Otitis media suppurativa chronica, left. Five weeks ago, ear dry. Since then, pain. Five days ago, facial paralysis, left. Four days ago polyp removed from ear. Left ear deaf. Spontaneous nystagmus in both directions. Caloric reaction positive.

Radical mastoid, left. Fistula in external semicircular canal. On probing, eyes deviate to opposite side. Two weeks later, vomiting. Increased nystagmus. Temperature, normal. Headache. Apathy. Next day, exitus.

Postmortem—Beginning meningitis. Abscess in left cerebellar hemisphere. Hydrocephalus internus.

Case 46. Milligan, W.[48]—Male, aged twenty-four years. Otitis media suppurativa chronica, right, for eighteen years. Three weeks before admission, headache and vomiting. Vertigo and constipation. Drowsiness. Temperature, 97° ; pulse, 56. Weber to right. Nystagmus to right. Optic neuritis. Kernig. Adiadokokinesis. Cerebrospinal fluid clear and under tension.

Operation. Radical mastoid. Cerebellum explored ; pus. Recovery.

Case 47. Milligan, W.[48]—Female, aged twenty years. Otitis media suppurativa chronica, right. Three weeks ago, headache and vomiting. Constipation. Coma. Temperature, 97° ; pulse, 60. Nystagmus to right. Optic neuritis. Kernig. Cerebrospinal fluid clear.

Operation. Radical mastoid. Cerebellum explored ; pus. Recovery.

Case 48. Mithoefer, W.[49]—Female, aged eleven years. Otitis media suppurativa chronica following measles, four years ago. On March 13, 1910, chill, fever and earache. Three days later, seen by writer. Small amount of foul pus. Perforation in Shrapnell's. Temperature, 101° ; pulse, 80. Headache and vomiting. Mastoid tenderness. No spontaneous nystagmus. Caloric positive. Next day, operation. Perisinus abscess. Sinus incised. Fifteen hours later, sudden cessation of respiration. Artificial respiration. Cerebellar dura in front of sinus incised and pus evacuated. Death.

Case 49. Mithoefer, W.[49]—Male, aged twenty-one years. Otitis media suppurativa chronica. Occasional earache. In December, 1908, pain was intense. Also chills and fever, and sweats. Tenderness over both mastoids.

Operation. Mastoid contained pus. Wall of sinus thick and gangrenous. Sinus incised. Free bleeding. Four weeks later, dizziness, nausea and vomiting. Had hearing. Temperature, 99.4° F; pulse, 56. Nystagmus to diseased side. Ataxia. Projectile vomiting. Inclination of head to diseased side. Optic neuritis both.

Operation. Cerebellar dura behind sinus incised; pus. Six days later, death, with signs of meningitis.

Case 50. Morton, J. P.[50]—Female, aged fifteen years. Mastoiditis, right. Temperature, 105° F. Mastoid operation. Three days later, temperature, 99°. Mental torpor. Patient lay on left side. No nystagmus. Right arm weaker than left. Vertigo. Tendency to fall. Exaggerated reflexes, right. Operation showed large cerebellar abscess. Meningitis. Exitus.

Case 51. Neumann, H.[52]—Male, aged thirty-five years. Otitis media suppurativa chronica, both, since childhood. Hearing in both ears, less in left. Weber to right. Radical mastoid, left. Dura in roof of antrum exposed; cholesteatoma in antrum. Prominence of external semicircular canal shows a black spot. Next day, April 7th, vertigo.

April 10—Headache. Temperature, 38.7° C. Improvement after changing dressing.

April 25—Headache, vertigo, fever. Pupils small and sluggish. Right optic neuritis. Rotatory nystagmus to left, especially on looking toward operated side. Diplopia, due to paresis of left sixth nerve.

April 26—Operation. External semicircular canal opened. Labyrinth contains pus. Nystagmus has changed. Is most marked on looking toward the right, and is almost horizontal. On looking to left, slight horizontal nystagmus to left. Somnolence. Headache.

May 4—Exitus.

Postmortem—Left sided cerebellar abscess. Suppurative labyrinthitis.

Case 52. Neumann, H.[52]—Female, aged thirty-four years. Otitis media suppurativa chronica, left. Six weeks ago, earache and deafness. Three weeks ago, headache. Polyp removed from left ear, followed by vertigo and diplopia, vomiting and unsteady gait. Objects seem to move to right. On examination, pulse 114. Intense headache. Head is turned to left. No rigidity of neck. Rotatory nystagmus to left, increased on looking to left, along with horizontal nystagmus to left. Left facial paresis. All reflexes increased. On standing with closed eyes, falls to right and backward. On rotating head, no difference. Walk unsteady. Left middle ear full of polypi. Pressure on polypi causes no dizziness. Weber to right. Rinné negative, left. No bone conduction.

May 14—Fetor ex ore. Vomiting. Operation. External semicircular canal rough. Sinus injured.

May 18—Rotatory nystagmus to left on looking to left. On looking to right, horizontal nystagmus to right.

March 23—Vomiting. Intense left-sided headache. Increased patellar reflex, both. Ankle clonus, left. Some rigidity of neck. Pulse, 132; respiration, 40. Lumbar puncture showed fluid under increased pressure, and turbid.

Operation. Exposure of cerebellar dura in front of sinus. A few drops of thick pus appear. Posterior surface of petrosa, including static labyrinth, removed. Pus in vestibule. Dura in region of saccus covered with granulations. Cerebellum incised in front of sinus; no pus. Sinus incised; no bleeding.

May 25—Vomiting. Pain in back of neck. Pulse, 84. Vertigo when lying on right side.

May 27—Exitus.

Postmortem—Abscess in left cerebellar hemisphere.

Case 53.—Neumann, H.[52]—Male, aged forty-five years. Otitis media suppurativa chronica, left, two and one-half years. For two days, intense headache, fever and chills. Vertigo for six weeks. Rotatory nystagmus to right, especially on looking to the right. Temperature, 39.8° C.; pulse, 102. Weber to right. Hears voice at one meter, left. Bone conduction diminished. Lumbar puncture; clear fluid; sterile. Radical mastoid operation. Large fistula in external semicircular canal. Perisinus abscess. Sinus thrombosed. Labyrinth opened; contained black masses. Sinus opened. Jugular ligated.

January 25—Facial paralysis, left. Nystagmus somewhat less.

February 3—Intense headache. On looking to right, nystagmus to right, as before. On looking to left, slight horizontal nystagmus.

February 4—Pulse, 69. Nystagmus has changed. It is most marked on looking to left, and is directed to left. Vomiting.

Operation. Removal of posterior surface of petrosa up to internal auditory meatus. Deep epidural abscess. Dura is sofe. Cerebellum incised in front of sinus; pus.

March 2—Operation. Incision through inner sinus wall. Second abscess found behind first.

March 13—Much improved.

March 31—Cured.

Case 54. Neumann, H.[52]—Female, aged twenty years. Admitted March 8, 1905. Otitis media suppurativa chronica, left, since seven years of age. Had operation on ear a year later. Since end of January, pain in left ear and profuse discharge. Since beginning of February, fever. On admission, fistula in mastoid. Weber to left. Rinné negative. Bone conduction diminished.

Operation. Radical mastoid; cholesteatoma. Dura of posterior fossa covered with granulations. Deep extradural abscess over posterior surface of petrosa. Cerebellum incised; pus. Sinus incised; thrombus. After operation, nystagmus to left only on looking to left. Vertigo.

March 11—On looking to left, nystagmus to left. On looking to right, very slight nystagmus to right. Empyema.

March 18—Exitus.

Postmortem—Abscess in left cerebellar hemisphere. Pial edema. Thrombophlebitis of left lateral sinus. Serofibrinous pleurisy, left. Compression of lung.

Case 55. Neumann, H.[52]—Male, aged seventeen years. Admitted April 27, 1905. Otitis media suppurativa chronica, right. Four years ago, fistula over mastoid, which healed. Four weeks ago, swelling over right mastoid. Headache. Emaciation. Vertigo. Vomiting. Speech is difficult. Tremor of right hand. Difficulty in walking. Arms and legs paretic and ataxic. No nystagmus. Neck tender. Scanning speech. Pulse, 60. Weber to left. Right Rinné negative. Air conduction much shortened.

Operation. Radical mastoid. Sinus exposed; cholesteatoma. Deep extradural abscess. Fistula in dura. Abscess in cerebellar hemisphere. Posterior part of labyrinth opened. Cochlea opened. After operation, no vomiting, no nystagmus. Facial paralysis, right.

April 28—Conjugate paralysis, right and upward. Headache. Patient lies always on left side.

May 2—Second abscess incised, followed by rotatory nystagmus to right.

Case 56. Neumann, H.[52]—Female, aged thirty-two years. Admitted April 2, 1901. Otitis media suppurativa chronica, right, eighteen years. For three weeks, earache. For one week, facial paralysis, right. Weber to right. Rinné negative, right. Hears whisper, right, at three meters.

April 3—Radical mastoid.

April 10—Headache. Irritability. Vertigo. Falls backward. Pulse, 64. Slight strabismus. Marked nystagmus, especially on looking to right. Reflexes increased. Ankle clonus. Eye grounds normal. Exitus in evening.

Postmortem—Old abscess in right cerebellar hemisphere. Pigmentation around abscess. Hole in dura, over region of ductus endolymphaticus. Deep epidural abscess of posterior fossa. Edema of brain. Acute hydrocephalus.

Case 57. Nourse, C.[53]—Male, aged six years. Otitis media suppurativa chronica, both, of eight months' duration. Pain in left ear five days. Headache. Vomiting. Mastoid tenderness. Mastoid operation. Perisinus abscess. Improvement. Sixteen days later, vomiting and headache. Temperature, 100° F. Drowsiness. Retraction of head. Exitus three days later.

Postmortem—Abscess in left lobe of cerebellum.

Case 58. Paterson, D. R.[54]—Female, aged fourteen years. Admitted February 27, 1907. Otitis media suppurativa chronica, left, following scarlet fever. Nine days before admission, headache. Temperature, 104°; pulse, 120. Right optic neuritis.

Operation. Cholesteatoma. Dura of middle fossa normal. Sinus apparently healthy.

March 13—Second operation. Cerebellum incised; pus. Cure.

Case 59. Paterson, D. R.[54]—Male, aged eighteen years. Admitted April 20, 1907. Five weeks before, pain in right ear. Headache. Vomiting. Diplopia. Drowsy. Temperature, subnormal; pulse, 54. Fetid discharge from right ear. Granulations. Nystagmus to both sides. Weakness of left external rectus. Double optic neuritis.

April 21—Operation. Cholesteatoma. Cerebellum and sinus uncovered. Cerebellum incised; pus. Cure.

Case 60. Paterson, D. R.[54]—Male, aged eleven years. Admitted March 21, 1907. Pain in left ear, headache, nausea and vomiting. Slight optic neuritis. Left membrana tympani red and bulging. No discharge.

April 4—Operation. Sinus in posterior part of bone, toward occiput, leading to fistula in dura. Incised. Cerebellar abscess found.

June 15—Discharged cured.

Case 61. Pierce, N. H.[55]—Female, aged twenty years. Seen February 7, 1909. Pain in right mastoid. Right facial paralysis. Earache began four weeks ago. Seven years ago, earache, right, and off and on since. Temperature, 99.5° F. Nystagmus to sound side. Vomiting. Neck rigid. Slight hearing, right. Answers slowly. Pulse, 60. Right disc blurred. Caloric negative. Labyrinthogenic cerebellar abscess was diagnosed.

Operation. Cholesteatoma. Fistula in external semicircular canal. Labyrinth opened. Cerebellar dura normal. Cerebellum explored behind sinus. Incision; pus. Hemiparesis of affected side was present after operation. It may have been there before operation. Cure.

Case 62. Rauzier and Roger.[56]—Male, aged twenty-four years. Admitted October 21, 1911. Otitis media suppurativa chronica, left. Headache, constipation, vertigo. Temperature, 39.2° C.; pulse, 68. Vomiting. Torpor. Lumbar puncture; cloudy fluid; sterile; under tension. Exitus.

Postmortem—Abscess in left cerebellar hemisphere. Mastoid full of pus.

Case 63. Rawling, L. B.[57]—Male, aged twenty-one years. Admitted December 1, 1905. Otitis media suppurativa acuta, left, four months. Three weeks ago, earache. Two weeks ago, frontal headache. Facial paralysis, left. Sways to left when standing with eyes shut. Temperature, 101° F.; pulse, 80.

December 5—Optic neuritis. Drowsiness.

December 7—Operation. Cholesteatoma. Improvement. Pulse, 64 to 88.

December 14—Trephine over cerebellum. Cerebellum incised; pus. Headache disappeared.

January 30—Discharged cured.

Case 64. Richards, J. D.[58]—Male, aged twenty-five years. Otitis media suppurativa chronica, left. On July 3, 1906. Schwartze-Stacke operation was done for the relief of otorrhea. Necrosis of tegmen tympani and posterior antral wall. Adjacent dura thickened and inflamed. On August 1, severe headache. Dizziness and vomiting. Tendency to fall to opposite side. Temperature, 97.8° F.; pulse, 50. Constipation. Reoperated. Epidural abscess above sinus knee evacuated. Next day, headache almost disappeared.

August 4—Papillitis. Next day, drowsiness and irritability. Answers delayed. Headache. Nausea. Emaciation. Temperature, 102.6° F.; pulse, 72 to 140. Horizontal nystagmus in both directions. Uvula deviates to right. Complete left facial paralysis came on a week after original operation. Sense of taste in anterior two-thirds of left half of tongue gone. Marked incoordination.

Operation. Incision through cerebellar dura one inch behind upper third of sinus; no pus. Incision internal to sinus, corresponding to posterior antral wall, going back one-half inch, evacuated large quantity of pus. Counter drainage from behind sinus. Recovery.

Case 65. Rutherford, H.[61]—Male, aged fourteen years. Headache and dizziness. Pulse, 52 to 62. Apathetic. Dusky and livid. Radical mastoid operation. Six days later, death from respiratory failure.

Postmortem—Cerebellar abscess. Granulation tissue around eighth nerve.

Case 66. Ruttin, E.[62]—Male, aged eighteen years. Admitted November 4, 1908. Periodic otorrhea, left, for eight years. Three to four weeks, pain and vertigo. Granulations and cholesteatoma, left. Has hearing. Weber to right. Rinné negative. Bone conduction shortened. No spontaneous nystagmus. On bending the head backward, nystagmus to right. Reversed fistula symptom. Caloric reaction positive. Temperature, normal.

November 10—Radical mastoid operation. Fistula in external semicircular canal. Next day, temperature 38° C. Headache. Nystagmus to sound side. Some rigidity of neck. Total deafness. Caloric negative.

November 13—Labyrinth operation. Escape of liquor. In the next six days, diminution of temperature. Pulse, 100. Nystagmus to sound side. On the seventh day, headache, slight nystagmus to diseased side, and vomiting. Temperature, normal; pulse, 70.

November 22—Nystagmus to left. Lumbar puncture; fluid clear. Pulse, 68. Cerebellum incised in region of posterior petrosal surface; pus.

November 21—Exitus.

Postmortem—Abscess in left cerebellar hemisphere, surrounded by edematous area. Hemorrhagic encephalitis. No meningitis. Pus showed streptococci.

Case 67. Ruttin, E.[62]—Male, aged sixty-eight years. Repeated otorrhea, left, for twenty years. Five months ago, swelling behind left ear. For two weeks, vertigo. Since day before, vomiting and headache. Has hearing. Nystagmus sometimes to right, sometimes to left. Slight fistula symptom. Caloric nystagmus lasts long time. Temperature, normal. Five days later, October 20, radical mastoid operation. Perisinus abscess. No fistula could be found in the labyrinthine capsule.

October 22—Marked nystagmus to right. Patient lies on right side. Completely deaf in left ear. Caloric positive. Vertigo.

October 25—Nystagmus to left. Ataxic gait. Vertigo. Eye grounds normal.

October 27—Sudden unconsciousness. Slight contractions of right hand. Pupils small, and do not react. Temperature, normal; pulse, 120.

October 27—Labyrinth operation, without narcosis. Incision of cerebellum over posterior petrosal surface; pus. Counter opening behind sinus. Conscious an hour later. No nystagmus. Five hours later, rotatory nystagmus to right. Babinski, left. Ataxia of left extremities.

October 29—Coma. Deviation of eyes to left. Cerebrospinal fluid cloudy.

October 30—Exitus.

Postmortem—Diffuse purulent meningitis. Cerebellar abscess, left. Streptococcus mucosus in cerebrospinal fluid and abscess.

Case 68. Ruttin, E.[62]—Male, aged eighteen years. Otitis media suppurativa chronica, left, following measles. Two weeks before, pain in ear.

February 11, 1910—Radical operation. Dura and sinus normal.

February 12—Temperature, 38.3° C. Vertigo. Nystagmus of first degree to sound side. Has hearing. Caloric negative.

February 21—Discharged.

February 23—Readmitted. Headache. Eye grounds normal. Temperature, 37.8° C. Labyrinth operation. Cerebellar dura near vestibule is discolered. Incision of cerebellum is negative.

February 25—Horizontal nystagmus to sound side. Rotatory nystagmus to diseased side. Vertical nystagmus on looking upward. Headache. Temperature, 36.7° C. to 37.2° C.; pulse, 68.

February 27—Pulse, 60. Diplopia on looking to left. Incision of cerebellum; pus.

March 13—Exitus.

Postmortem—Hemorrhagic abscess of left cerebellar hemisphere. Circumscribed lepto- and pachymeningitis interna on posterior surface of petrosa. Internal hydrocephalus. Abscess contains streptococcus pyogenes. Fluid in lateral ventricles sterile.

Case 69. Ruttin, E.[62]—Male, aged fifteen years. Admitted December 18, 1909. Otitis media suppurativa chronica, right, for three years. Had polyps removed several times; the last time being three days ago. A week ago, headache. After the polyp extraction, fever and chills. On admission, temperature, 38.4° C. Had vertigo for a long time. Nystagmus to both sides. Right ear is deaf. Rinné negative. Caloric negative. Pulse, 104. Slight rigidity of the neck. Dermographia. Kernig.

December 17—Operation. Pus and cholesteatoma. Perisinus abscess. Dura of middle fossa shows yellowish discoloration. Incision of sinus; thrombus. Jugular ligation. Incision of temporosphenoidal lobe negative. Incision of cerebellum in front of sinus; pus.

December 18—Paralysis of movement of eyes to right. Rotatory nystagmus to left. Slight facial paresis. Rigidity of neck. Knee jerk absent, right. Present on left side. Kernig.

December 19—Rotatory nystagmus to right, and vertical. Eye grounds normal.

December 24—Rotatory nystagmus, right and left. General hyperesthesia. Dermographia. Ataxia of upper and lower extremities.

December 28—No ocular paralyses.

January 4—Completely deaf, right.

January 20—Cured.

Case 70. Ruttin, E.[62]—Male, aged twenty-one years. Admitted July 31, 1910. Otitis media suppurativa chronica, right, ten years. For past eight years attacks of vertigo. For one week headache and vomiting. Right ear deaf. Caloric negative. Slight facial paresis, right. Temperature, 38.5° C.; pulse, 76. Kernig. Eye grounds normal.

July 31—Operation. Cholesteatoma. Fistula in external semicircular canal. Facial exposed in horizontal and descending portions. Stapes missing. Labyrinth operation. Lumbar puncture gives cloudy fluid.

August 1—Rotatory nystagmus, left. Headache.

August 3—Feels better. Pulse, 56.

August 6—No nystagmus. Feels well. Ataxia in both upper extremities, not in lower extremities.

August 10—Headache. Rotatory nystagmus right and left. Apathy. Pulse, 60. Incision of cerebellum, below sinus; pus.

August 11—No nystagmus or headache. Cerebellar pus shows neither pus cells nor bacteria. Sediment consists of detritus.

August 12—Rotatory nystagmus to left and to right.

August 30—Discharged.

October 4—Readmitted. Headache. Vertigo. Pulse, 68. Horizontal nystagmus, right and left.

October 5—Operation. Below first abscess another abscess was found. Contains streptococci and anaerobic bacilli.

October 7—Improvement.

December 21—Discharged cured.

Case 71. Ruttin, E.[62]—Male, aged forty-seven years. Admitted February 8, 1911. Two years ago struck by lightning. Since then deaf in left ear. Three weeks ago, pain and discharge from left ear. Has some hearing. Rinné negative. Bone conduction diminished. No spontaneous nystagmus. Fistula test negative. Rotation, twenty-four seconds, both sides.

February 14—Vomiting and vertigo. Headache. Rotatory nystagmus, right and left. Temperature, normal.

March 7—Operation. Cholesteatoma. Facial lies uncovered. Dura of both fossæ exposed and covered by cholesteatomatous membrane.

March 8—Rotatory nystagmus, right. Facial paralysis.

March 13—Caloric negative. Completely deaf. Rotatory nystagmus, right and left. Bárány's pointing tests typical.

Labyrinth operation. Incision of cerebellum; pus.

March 15—Coma.

March 17—Exitus.

Postmortem—Purulent basilar meningitis. Abscess of left cerebellar hemisphere. Pus from abscess contains staphylococcus pyogenes.

Case 72. Ruttin, E.[62]—Male, aged twenty-three years. Admitted July 1, 1910. Otitis media suppurativa chronica, left, after scarlatina. For two weeks, headache and vertigo. No fever. For two weeks, facial paralysis, left. Has hearing. Rinné negative. Bone conduction increased. No spontaneous nystagmus. Caloric positive.

July 4—Radical operation.

July 7—Vertigo.

July 15—Vomiting. Headache. Temperature, 38° C. Spontaneous nystagmus to both sides. Caloric negative. Rotation test. right thirty seconds; left, nothing. Hearing is questionable.

July 19—Chill. Operation. Stapes absent. Probe falls into oval window without resistance. Labyrinth operation.

July 21—Feels well. Rotatory nystagmus, right and left. Pyemic temperature curve.

July 26—Jugular ligation. Thrombus removed from sinus.

July 29—Edema of right conjunctiva.

July 31—Exophthalmos, right.

August 2—Exitus.

Postmortem—Purulent basilar leptomeningitis interna. Abscess on under surface of left cerebellar hemisphere. Thrombophlebitis of left sigmoid sinus, jugular bulb, inferior petrosal sinus and both cavernous sinuses.

Case 73. Sack, N.[63]—Female, aged five years. Admitted August 31, 1907. Otitis media suppurativa chronica, left, for two years. Two weeks ago facial paralysis, left. Somnolence. Pain in left ear. Temperature, normal; pulse, 115. Vomiting. Horizontal nystagmus to left. Patient falls to left. Ataxic gait.

September 6—Radical mastoid operation. Sequestrum involving part of the cochlea and semicircular canals removed. After operation pulse dropped to 64 to 62. Nystagmus changed to right. Exitus four days later.

Postmortem—Two abscesses in left cerebellar hemisphere. Necrotic area on posterior surface of petrosa, in region of saccus. Pus from abscess showed Vincent's bacillus and spirilla.

Case 74. Savariaud and Dutheitlet de Lamothe.[64]—Male, aged fourteen years. Otitis media suppurativa chronica, right. Admitted March 3, 1911. Headache, vertigo and vomiting. Vomiting is of cerebral type. Apathy. Temperature, 37° C.; pulse, 52. No mastoid symptoms. No Kernig. No rigidity of neck. Slight inequality of pupils; react normally. Slight papilledema. Walks easily. No asynergy or unmeasured movements. Romberg. On lying down there is some imprecision of movements of right side. No adiadokokinesis. Lumbar puncture shows slightly turbid fluid; sterile

March 4—Operation. Radical mastoid.

March 5—Second operation. Cerebellar dura incised. Cerebellum herniates into wound. Incised; pus. Some improvement. On third day signs of meningitis. Exitus.

Postmortem—Purulent leptomeningitis. Cerebellar abscess. Fistula leading from antrum through posterior surface of petrosa to cerebellum.

Case 75. Scott, S.[66]—Male, aged twenty-eight years. Admitted July 15, 1908. Otitis media suppurativa chronica, left. Headache. Deafness, left, three years and nine months ago. Vertigo, vomiting and headache. Unable to walk straight. Hearing by bone conduction, in left ear, completely lost. Romberg. Falls to right. On walking swerves to right. Spontaneous nystagmus to left. Slight nystagmus to right on looking to right. Eye grounds normal. Kernig. Temperature, 98° F.; pulse, 72. Lumbar puncture showed sterile fluid, not under pressure.

Radical mastoid operation. Large fistula in semicircular canal region. Facial nerve exposed and surrounded by granulations. Granulations in cochlea and vestibule. Labyrinth operation. Granulations on cerebellar dura, mesial to sinus. Incised; pus. Pulse rate became slower—52. Incoordinate movements of left upper extremity.

Second operation. Dural incision enlarged. A large cerebellar hernia formed. Facial paralysis, left. Gradual improvement of symptoms.

Case 76. Spencer, F. R.[67]—Male, aged nineteen years. Seen January 13, 1910. Earache, left, for two days. Otitis media suppurativa chronica since childhood. Mastoid tenderness.

On January 19, simple mastoid operation. Sinus exposed. Was yellow, with no pulsation. Incised, and thrombus removed. Jugular was not tied. Pus had a fecal odor. Patient discharged in three weeks. Four days later, intense pain in left side of head. Sometimes on right side. Vertigo and occasional vomiting, without nausea. Drowsiness. Rapid emaciation. Sallowness. Temporosphenoidal lobe and cerebellum were explored. No pus found. Death four days later. Pus from mastoid and lateral sinus showed pneumococcus and an organism from the paracolon group.

Postmortem—Cerebellar abscess in left hemisphere.

Case 77. Texier, V.[68]—Male, aged thirty-one years. Admitted February 13, 1908. Otitis media suppurativa chronica, left. Vertigo and vomiting. Tendency to fall to right. Lumbar puncture shows normal fluid. Temperature, normal. Caloric positive. Rotation test normal.

Radical mastoid operation. Some erosion of external semicircular canal, but no fistula. Vertigo and vomiting continued. Headache. External semicircular canal opened; no pus. Ataxic gait. Scanning speech. Cerebellum explored. Patient succumbed to anesthesia.

Postmortem—Large cyst in left cerebellar hemisphere.

Case 78. Thompson, J. A.[70]—Female. Otitis media suppurativa chronica, both. Had hearing. Radical mastoid operation, right. Cholesteatoma.

Postmortem—Suppurative labyrinthitis, right. Extension along auditory nerve. Abscess in right cerebellar hemisphere. Suppurative labyrinthitis, left. Abscess in left cerebellar hemisphere.

Case 79. Thompson, J. A.[70]—Otitis media suppurativa chronica, left, for thirty-five years. Acute exacerbation five weeks ago, with nausea, vomiting, vertigo and intense headache. Temperature, 103° F. Facial paralysis in the fifth week.

Radical mastoid operation. Seven days later cerebellum incised, and abscess found behind internal aditory meatus. Exitus next day.

Case 80. Turner, A. Logan, and Reynolds, F. M.[71]—Male, aged twenty-six years. Otitis media suppurativa chronica, left. Symptoms of sinus thrombosis. Sinus opened and jugular ligated. Cerebellum explored behind sinus. No pus found. Recovery.

Readmitted four months later. Pulse and temperature normal. Pulse under 60 several times. Marked deafness. Weber to right. Schwabach shortened. Spontaneous horizontal nystagmus to right. Caloric negative. Slight Romberg. Cerebrospinal fluid normal.

Second operation. Cholesteatoma. Fifteen days later, vomiting. Pulse, 54. At five o'clock in the evening, sudden death.

Postmortem—Large abscess in left lobe of cerebellum. Some edema of brain. No meningitis. Pus showed streptococcus pyogenes and bacillus proteus. Examination of labyrinth showed subacute inflammation with tendency to connective tissue formation. No pus.

Case 81. Uffenorde, W., and Weber, L. W.[72]—Male, aged nineteen years. Otitis media chronica subacute, right, with serous exudate. On July 21st, congestion of membrana tympani on left side. Temperature, 39.5° C.; pulse, 110. Next day some mastoid tenderness and slight rigidity of neck.

July 25—Simple mastoid operation. Pus shows streptococcus. Headache for next two days.

July 28—Radical mastoid operation. Anterior half of external semicircular canal looks blackish. Dura of middle fossa exposed. Looks normal. Next day vomiting. No headache. Temperature, 38° to 39° C. No nystagmus or vertigo.

July 31—Exploration of sinus. On pressing sinus pus exudes from emissary. Ligation of jugular. Sinus split and thrombus removed. Patient felt well until August 16th, when suddenly vision became very poor. Congestion of retinal veins. Counts fingers at two or three meters. Next day, pupils dilated. Marked choked disc, both sides. Star shaped figure in left macule, like in albuminuric retinitis.

August 18—Incision of cerebellar dura evacuated bloody cerebrospinal fluid containing pus. Subcortical cavity in cerebellum. Drainage.

August 23—Macular figure diminishing. Vision, three to four meters.

August 27—Sensorium normal. No ataxia. No disturbances of motility or sensation of upper extremities. Tongue deviates to left. Slight ptosis of left upper lid. Reflexes up.

September 15—Attack of Jacksonian epilepsy, beginning in left leg.

November 4—Discharged cured.

November 5—Examination by Dr. Weber. Tongue deviates to left. Right thigh and leg four centimeters thinner than left. Right foot in varoequinus position. Right side, Babinski. Left patellar and ankle clonus. No vertigo or ataxia. The spastic paresis and Babinski of the right leg indicate a lesion of the left pyramidal tract above the decussation. Deviation of tongue to left also points to this. Due to inflammation on under surface of left cerebellar hemisphere, causing pressure on pyramidal tract.

Case 82. Uffenorde, W.[73]—Male, seventeen years. Otitis media suppurativa chronica, right. For past ten days, weakness, irritability, and headache. For past week, discharge stopped. Unsteady gait. Tenderness over mastoid. Functional tests, right ear: No bone conduction; Weber is contralateralized; Rinné negative. Left, normal. Gait is uncertain. With eyes closed deviates to right. Temperature, 39.5° C. Irregular nystagmus. Right sided choked disc.

April 10, 1906—Operation. Pus in antrum. Cholesteatoma. Granulations over dura of posterior fossa. Sinus incised; no bleeding. Cerebellum incised; no pus. Improvement in symptoms.

May 1—Plastic.

May 21—Headache, nausea, vomiting, vertigo. Eye grounds normal. Pulse, 80. Falls toward diseased side.

May 25—Temperature, 35.7° to 37.6° C.; pulse, 60 to 68. Babinski both sides. Marked nystagmus toward diseased side. Slight nystagmus toward opposite side.

Operation. Incision of cerebellum; pus. Glass drain.

July 8—Discharged cured.

Case 83. Uffenorde, W.[74]—Male, aged five years. Five weeks ago, scarlet and diphtheria. Four weeks ago, otitis media suppurativa acuta, both. Irregular temperature; vomited twice. Apathy.

April 13, 1909—Operation, left side. Perisinus abscess. Epidural abscess on posterior surface of petrosa. Stapes is wanting. Probe passed into vestibule without obstruction. On withdrawal, pus follows. Fistula in upper part of promontory. Labyrinth operation. Saccus endolymphaticus is plainly seen in dura. It is ruptured.

April 17—Vomiting. Lumbar puncture. Cerebrospinal fluid cloudy. Streptococcus in pure culture. Puncture of cerebellum through inner wall of saccus; pus. Glass drain.

April 21—Exitus.

Case 84. Van den Wildenberg.[75]—Female, aged twenty-seven years. Admitted June 14, 1908. Otitis media suppurativa chronica, right. Periodic exacerbations. Frequent attacks of headache. Vertigo. For past two weeks, intense pain in right ear. Chills. On admission, temperature, 41° C.; pulse, feeble and rapid. Tenderness over emissary. Tenderness over right side of occiput. Swelling along right jugular. Cerebrospinal fluid normal.

June 15—Operation. Jugular resection. Radical mastoid. Perisinus abscess. Sinus incised; thrombus.

June 16—Temperature, 38° C. Slight headache, vertigo. Patient has hearing.

June 19—Bulb operation. Contains broken down clot.

June 20—Slight facial paresis. Pain in chest. Râles. Emaciation.

June 21—Vertigo. Ataxic gait. Falls toward diseased side. No nystagmus. Pain and tenderness in occipital region.

June 24—Cerebellum incised; pus.

September 10—Discharged cured.

Case 85. Venin, M.[76]—Male, aged nineteen years. Otitis media suppurativa acuta, both. Left side got worse. Walking became difficult. Vomiting, headache. Entered hospital, August 16, 1910. Remission of symptoms.

September 4—Intense headache. Slight weakness on left side. On walking, tendency to deviate to right. Photophobia. Horizontal nystagmus to right only on looking to extreme right. Slight bilateral congestion of optic nerves. Pulse, 56.

September 6—Operation, left. Sinus and dura of posterior fossa exposed. Appear normal. Two punctures of cerebellum. No pus.

September 8—Cerebellum again incised; pus. Cure.

Case 86. Whitehead, A. L.[78]—Male, aged fifteen years. Otitis media suppurativa chronica, left. Two weeks, pain and mastoid swelling. Temperature, 99° F.; pulse, 98. Radical mastoid operation. Perisinus abscess. Persistent headache. Two days later, optic neuritis. Nystagmus on looking to left. Cerebellum explored; pus. Recovery.

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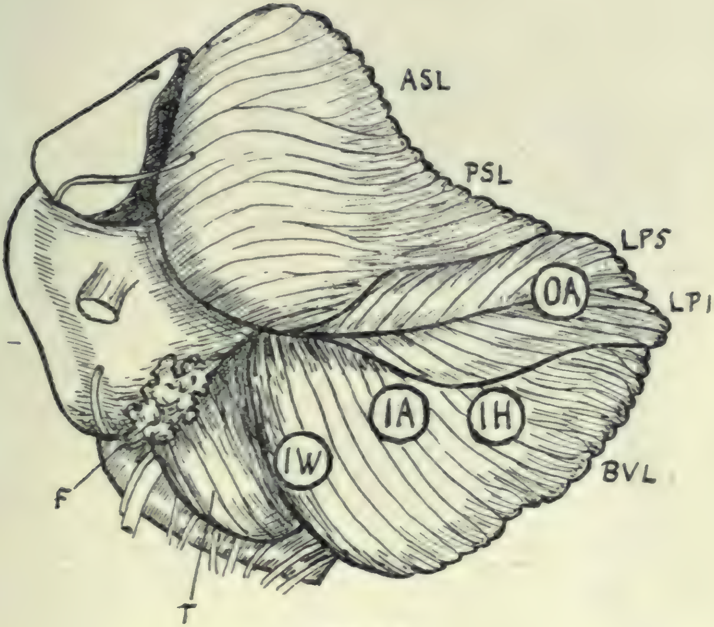

FIGURE 1.

Lateral View of Cerebellum.

OA—Center for outward movement of arm.
IA—Center for inward movement of arm.
IW—Center for inward movement at wrist.
IH—Center for inward movement at hip.
ASL—Anterior semilunar lobe.
PSL—Posterior semilunar lobe.
LPS—Posterosuperior lobe.
LPI—Posteroinferior lobe.
BVL—Biventral lobe.
T—Tonsil.
F—Flocculus.

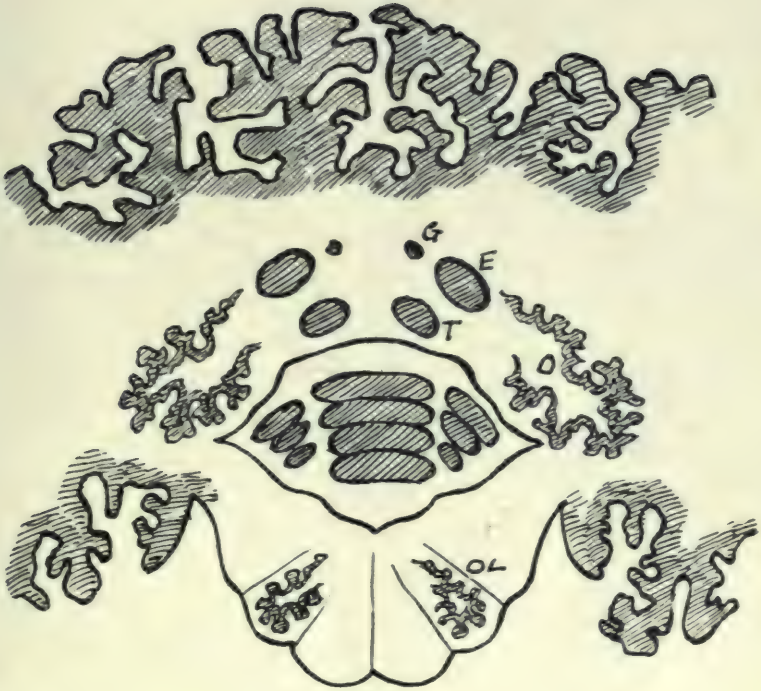

FIGURE 2.

Transverse Vertical Section Through Cerebellum and Medulla.

O—Cerebellar olive.
T—Nucleus tecti.
E—Nucleus emboliformis.
G—Nucleus globosus.
OL—Medullary olive.

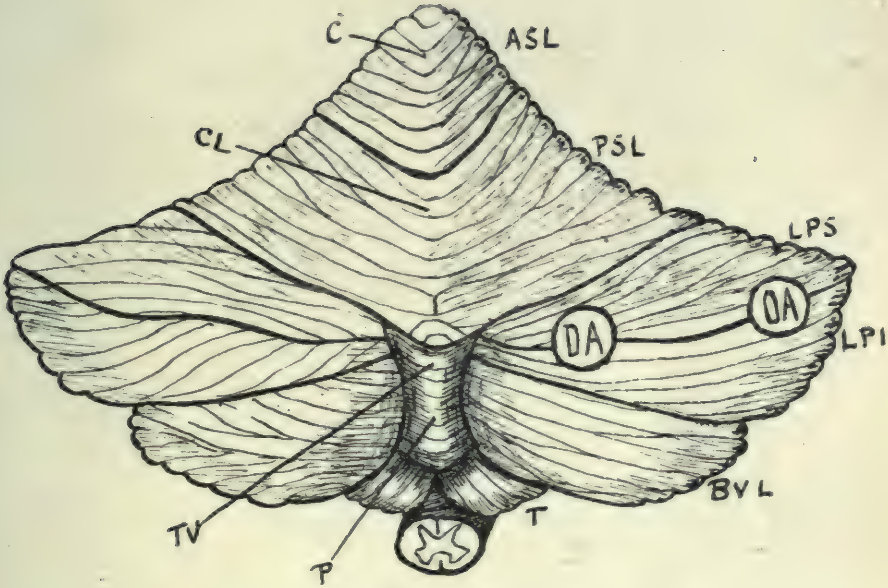

FIGURE 3.

View of Cerebellum From Behind.

OA—Center for outward movement of the arm.
DA—Center for downward movement of the arm.
ASL—Anterior semilunar lobule.
PSL—Posterior semilunar lobule.
LPS—Posterosuperior lobe.
LPI—Posteroinferior lobe.
BVL—Biventral lobe.
T—Tonsil.
P—Pyramid.
TV—Tuber valvulae.
CL—Clivus.
C—Culmen.

II.

SOME REMINISCENCES, REFLECTIONS AND CONFESSIONS OF A LARYNGOLOGIST.*

By John Noland Mackenzie, M. D.,

Baltimore.

In responding to the courteous and flattering invitation to be with you this evening, and in casting about for a subject upon which to address you, it has occurred to me that, instead of treating you to the arid narrative or perhaps profitless discussion of some of the unsettled questions which have divided laryngologic opinion now and at other times, and upon which I, and not you, should seek enlightment, I would ask you to come and live with me for a few moments in another and an earlier time, in a distant day and atmosphere, in which the grown-up virile puissant laryngology of this great age of human progress was yet in its swaddling clothes. And if in doing so I shall become personal as well as reminiscent, shall give you a chapter out of my own life and my own experience, I pray you to be charitable enough not to misunderstand me, but let me believe that my words are not addressed to the unsympathetic and critical ear of strangers, but to a little circle of friends gathered around the family fireside, where I may talk with the freedom of one who is at home, and where I feel that to many of you at least I may speak, as the ancient Roman has it, "in loco parentis."

In the seventies and early eighties of the last century, the Hospital for Diseases of the Throat and Chest in Golden Square, London, was the Mecca of the vast majority of English-speaking students of laryngology, who came there attracted by the reputation and engaging personality of Morell Mackenzie, then at the zenith of his professional career. Whether they went for study elsewhere or not, sooner or later their footsteps turned to Golden Square, either for passing curious observation or more serious and continuous work. The institution itself was a model of simplicity, both in architecture and equipment. In secluded isolation, it stood in the little square in the narrow zone which separates the throbbing, restless, rushing life of the metropolis from the poverty and squalor of the slums, and in a silence broken only in the daytime by the roar of Regent street, near by. The lower floor of the building, where most of the work of the hospital was done, consisted of a very large waiting room. It had to be large to accommodate the then largest laryngologic clinic in the world, an examination room of ample size, but simply furnished, which, in turn, opened into a very much smaller and more private one, which was consecrated mainly to physical diagnosis. In the more capacious apartment were tables and lamps for the attending surgeons and chief of staff. The upper floor was occupied by the in-patients, and was always full. It was amid these modest surroundings, in picturesque contrast to the princely apartments and imposing apparatus of some of our modern, up-to-date laryngologic establishments, that many of the men who were destined to direct the after-progress of laryngology in Great Britain and America were not taught, but learned for themselves, the first lessons of their art. I entered on my duties as chief of staff in the summer of 1879. My predecessors in office were Felix Semon, Samuel Johnston of Baltimore and Lennox Browne. I had qualified beforehand for the job, and having acquired the principia of the subject in the old Metropolitan Throat Hospital in New York, in spare hours snatched from my service in Bellevue Hospital, and under the tutelage and inspiring example of my old friend, Clinton Wagner, one of the pioneers of the specialty in America and founder of the New York Laryngological Society. At that time, with all the enormous material at our command, there was practically no instruction given, except in the way of hasty demonstration of cases, and if the student or visitor learned anything, it was through close personal observation on his part, and not through any gigantic effort to impart knowledge on the part of the medical staff. With one or two exceptions, the latter directed their attention almost solely to the larynx and thyroid gland, and the nasal passages were only examined when in quest of a polypus or when the attention was irresistibly attracted to these organs by the horrible stench of an ozena. The nasal cavities were practically neglected, and the only apparatus in the hospital for the treatment of their diseases consisted of a pair of forceps for the removal of nasal polypi, and a handball atomizer with a detergent solution for the treatment of ozena or any other miscellaneous disease of the nose that might irresistibly obtrude itself upon the recognition of the medical staff.

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*Address delivered at the College of Physicians, Philadelphia, at a meeting of the Philadelphia Laryngological Society, March 7, 1916.

I have said that we concerned ourselves chiefly with affections of the larynx and thyroid gland. Let me pass briefly in review our then treatment of some of these affections. Among our most frequent visitors to the clinic were cases with tuberculosis of the larynx. They literally abounded. They came by the hundreds, in striking and conspicuous contrast to the comparative rarity of this disease in the throat clinics of today. Many of them presented the classical picture first drawn by Morell Mackenzie, which we considered then pathognomonic of tuberculosis. It should be stated here that the grouping of signs, turban epiglottis, pyriform aryepiglottic folds, etc., so graphically described by Mackenzie, was not considered characteristic outside of England, and I must say that in my own observation I have never seen such constancy in the ensemble or grouping of appearances, either on the Continent or in the United States. At that time much difference existed among laryngologists concerning the value of laryngoscopic diagnosis in this disease. Ziemssen was the first to deny its certainty, and subsequently Heinze, and even Morell Mackenzie, gave in their adhesion to his views. Lennox Browne, on the other hand, goes to the other extreme, or, rather, went to the other extreme, and declared that, with the exception of laryngeal growths, there is no disease of the larynx in which we may be so sure of laryngoscopic diagnosis. The partisans of both these views are too sweeping in their statements. There are many cases met with in practice in which the diagnosis must remain in doubt. I believe the case can be briefly stated as follows: The diagnosis of the lenticular ulcer, especially when single or unassociated with other tubercular lesions, is laryngoscopically impossible; even when the ulcers are multiple, and bilaterally situated on the cords or vocal processes, then can only be looked upon with suspicion in the absence of other signs of the disease. This same is true of the aphthous or diphtheritic ulcer. Even the ulceration which results from infiltration is not always typical in appearance, and may need the association of other tubercular lesions to proclaim its true nature. When infiltration is unilateral, the diagnosis is sometimes shrouded in doubt, except when multiple ulceration has taken place. The association of the turban-shaped epiglottis with the pyriform swelling of the aryepiglottic folds is characteristic of tuberculosis, and should not be confounded with edema, its closest simulator. It should here be remembered, however, that edema may complicate the case and cause uncertainty as to its real nature. When to the characteristic infiltration of the epiglottis and aryepiglottic folds is added the typical worm-eaten superficial serpiginous ulceration, the grouping in the picture is pathognomonic. The typical laryngeal tubercular ulcer resulting from infiltration, in its development, appearance and course does not resemble any other laryngeal disease with which I am familiar. The lesions of this—and this is true of many other diseases—may, of course, occur in atypical forms, the characteristic grouping may not be present, and the diagnosis may be left for a while in doubt, but this does not invalidate the position that tuberculosis manifests itself laryngoscopically (in the larynx) in a manner different from any other known form of disease.

The subject may seem trite and superfluous, but I have introduced these remarks on the laryngeal picture in tuberculosis in order to emphasize the importance of the laryngoscopic or naked eye method of diagnosis (supplemented or not, as the case may be, by clinical phenomena) to the exclusion, if possible, of the microscope and the laboratory in the detection of disease in the larynx. In these days of unquestioning reliance on and faith in chemical and other strictly scientific, as contradistinguished from purely clinical aids in disease discovery, it is in many quarters at least fast becoming a lost art. As the introduction of the modern, direct methods of examination have done away largely with much of the old-time manual dexterity in intralaryngeal and tracheal manipulation, so the common use of laboratory tests have, by opening up a lazier and easier road to diagnosis, greatly dulled the former diagnostic sense and diagnostic acumen. To overcome this unfortunate condition of affairs in the coming generation, the student should be shown as many cases as possible of a given malady, in order that the picture of the disease may be so photographed on his brain and embedded in his memory that he may be able to recognize it by sight alone when brought before its image in the mirror. Take him back to the old-time initiative in diagnosis, teach him to rely more upon his special senses, tell him that, after all, personal observation and clinical experience are less fallacious than the more artificial, although more strictly scientific methods, even though the latter may be, in many cases, indispensable and in some ways more exact. It is only human to make mistakes. Let him not, therefore, in case of doubt, run to the laboratory at once for help, as is the rule in most cases today, but without in the least degree underestimating its inestimable value and assistance, let him seek it as the court of last resort. In other words, let him make the diagnosis with the naked eye alone; form his opinion in this way first, even though he may have to control it in the end by scientific tests.

The student should be schooled not only in the naked-eye appearance of laryngeal disease, but also in what is even more necessary—early laryngoscopic diagnosis. The supreme and far-reaching importance of the latter ought to be evident to intuition, and the very mention of the subject in a company of laryngologists should be looked upon as a piece of gross impertinence: and yet, unthinkable and incredible as it may seem, to many workers in this special field today, whose vision does not carry beyond the tonsil, whose horizon is the palatal arcade and whose ignorance of what lies beyond it is as boundless as the deep, it is a neglected or even unknown accomplishment. A fellow-laryngologist, an excellent man and a good surgeon, who had wasted a number of years out of what might have been a wholly useful life removing tonsils and doing submucous resection, when asked by a brother practitioner about the laryngeal appearance in one of his cases, exclaimed. "Larynx! I know nothing about the larynx!"

It is impossible to exaggerate the importance of the laryngoscope to the medical and surgical diagnostician in the early detection of disease, not only in the respiratory organs themselves, but, of equal, if not superior importance, in neighboring and remote organs of the body. Long in advance of the appearance of classical signs and symptoms of disease in other organs, it often points the way to grave disorder.

Not to multiply examples, how often is such a seemingly innocent performance as a lame or staggering cord in an otherwise apparently healthy individual the early herald of the existence of some formidable affection, such as malignant growth, central nerve disease or aneurism, or a tiny moist or weeping ulcer on the vocal process or a vegetation in the interarytenoid space to the alert and practiced eye the tell-tale, though silent, witness of tuberculosis!

Every tumor of the larynx, no matter how benign it may appear, should be examined with the greatest possible care. Some of the most fatal diseases known to man make their first appearance in the larynx in the guise of great benignity. Thus the presence of cancer and tuberculosis in the individual is often first proclaimed by the discovery of an apparently simple papillomatous excrescence in the larynx. By the careful study of every case coming under our observation we will some day, among other things, clear up the mystery which surrounds the genesis of papilloma and approach more closely with the naked eye alone the earliest possible recognition of some of the most deadly diseases of the larynx.

One day a man was admitted to my division in Bellevue Hospital with the diagnosis of typhoid fever, but who presented an appearance which I had never seen in that disease. Not knowing what ailed him, I called the resident medical staff in consultation, and the consensus of opinion was that, while his trouble was not such as had been diagnosticated, it was impossible to classify it, to give it a name, as none of us had ever seen its like before. Janeway was summoned. "Acute ulcerative endocarditis." In view of the fact that there were no detectable morbid heart sounds present, the diagnosis was a brilliant one, and was verified by autopsy on the following day. At that time the disease was practically unknown, certainly not generally known, on this side of the Atlantic. I had read somewhere an abstracted account of the affection, taken from a French journal, but had forgotten it. In the absence, apparently, of heart trouble, how could you possibly make such a diagnosis, he was asked. "I don't know, but I have seen a case," was his reply.

The local treatment (of laryngeal tuberculosis) consisted in the use of hot and cold soothing and stimulating inhalations, the insufflation of an opiate, generally morphin, followed by a mixture of iodoform and starch and the use of the esophageal tube to facilitate deglutition, and our one consoling thought was that we had placed our patient in a condition in which he would suffer the minimum amount of pain and approach the end, if possible, with resignation and an equal mind. All were doomed to die; the appearance of the disease in the larynx was the warrant of death. We did not know then that some of the very worst cases could be saved. It was not until years after that I had the truth of this latter statement brought forcibly home to me by the following case, which I take from several others quite as remarkable:

Mr. B., a small, important little man, with an iron will, came from a distant city to consult me before going west for his health. He had a good-sized cavity in the left apex; both ventricular bands were the seat of broken-down infiltration, the ulceration covering both surfaces completely and extending into the ventricles. There was marked infiltration of the ary-epiglottic folds (pyriform swelling), which had, however, not yet ulcerated. Physically, he was in very bad shape; was very weak, and in no condition to undertake even a short journey. I advised him to return to his home, give up his business (that of banker) and live in the open air, and on no account to attempt his trip to the West, as he might never reach his destination alive. It was Seneca who said that it is a part of the cure to wish to be cured. Well, this little man wished to be cured, and cured at all hazards. And he therefore did not take my advice. The next time I saw him, two years later, his chest cavity had become obsolete, the laryngeal infiltration had disappeared, and the ulceration had completely healed. He had gone to Colorado, pitched his tent in the wilderness, lived in the open air, and, as far as possible, in silence and in solitude, and during the entire absence from home had taken no medicine, nor had he even laid eyes on a physician. Again, and against my advice, he returned to his former business. In six months ulceration broke out afresh in the larynx. This time I told him to take the fastest express to the West. To make a long story short, I saw him twenty-one years after his first visit to me. He was perfectly well, and, apart from the scars in the larynx, no one would have known that he had ever been hurt. Remember, this was not a case which happened yesterday, or the day before, but long years before man had surrendered to nature the care of tuberculous disease.

Among the cases of general and laryngeal tuberculosis which presented themselves for treatment there were a goodly company of the variety of laryngeal neoplasm, which I have called in my classification of the laryngotracheal neoplasms occurring in that disease, papillomatous excrescences, vegetations and tumors, and which I have discussed at length elsewhere.[1]

As this form of tumor is yet imperfectly understood, as very little is still known concerning its histologic nature, and in view of the fact that the determination of its essential character will have an important bearing on its treatment, I will again venture a word concerning it. These are the members of the second group, and are closely allied macroscopically to simple laryngeal papillomata, for which they are easily mistaken. They are the growths which every student learns to recognize in the alphabet of his special studies. They are often the avant courrier of laryngeal and pulmonary tuberculosis, and may remain for a long time as the solitary outward and visible sign of that disease. Their presence in the interarytenoid fold often furnishes strong presumptive evidence of incipient tuberculosis. They vary greatly in size, shape and situation, sometimes projecting from under the anterior commissure of the larynx in the form and appearance of a spray of coral, at others filling the larynx with growths macroscopically indistinguishable from simple papillomata, which are sometimes so abundant as to cause stenosis and necessitate tracheotomy. Their most characteristic seat is the posterior laryngeal wall, where they appear as warty acuminate or leaflike outgrowths of a pale grayish or pronounced reddish hue, or banked at that situation in a solid mound, either smooth in contour or bristling with multiple acuminate projections. The histology of this class of tumor has been imperfectly studied, and may well in the future bear a more careful scrutiny. Stoerck, who first called attention to their presence in the interarytenoid fold as an infallible sign of incipient tuberculosis, following Rokitansky, regarded it as the result of an indurated proliferation of the connective tissue which occurs in the course of chronic tubercular disease of the mucous membrane in the neighborhood of the arytenoid cartilages. Kundrat, who examined several of Stoerck's cases, pronounced them essentially papillomata and nontubercular in origin. I know of no other special observation on the subject, with the exception of the microscopic appearances of a case of my own, examined for me by Sydney Cone, then pathologist to the surgical department of the Johns Hopkins Hospital, many years ago, and reported to the otolaryngologic section of this college in December, 1904. I have not time to go into a detailed account of the anatomic report. Suffice it to say that in the sections examined the picture was that of a tuberculosis of a papilloma—papillomatous tissue infected with tubercular tissue. Whether or not the growth is originally tubercular or becomes so secondarily through infection is a point to be determined by further observation. Without going into explanatory detail, the study of the sections developed facts which were not only of histologic, but also of eminently practical importance. It was especially interesting from a diagnostic point of view in the microscopic differentiation of this form of outgrowth from the papillary variety of epithelioma, particularly when, as sometimes happens, the tubercle bacillus is found only after a diligent and prolonged search. It also went to show that incomplete attempts at removal only served to stimulate the local growth of the neoplasm and increase the danger of reinfection. It will be the task of the future to determine whether all growths of this nature found in a tubercular subject show a tubercular structure, or whether there are some that adhere to the strictly papillomatous type. Whether benign or tubercular, the very fact that this variety of tumor often heralds the approach or proclaims the presence of tuberculosis in the individual only emphasizes the importance of examining with care not only clinically, but microscopically, all papillomata taken from the larynx and trachea. With regard to their mode of development, it is quite possible, as Cone has suggested, that in some cases at least they may have an origin analogous to the papillomata found in the urethra and vagina, which are probably produced by infection by the tuberculous discharge from the bladder and uterus.

This variety of outgrowth was always looked upon as perfectly legitimate surgical prey. I have often removed the entire tumor or portions of it, calmly and utterly innocently oblivious of the fact that I was in so doing stimulating its local growth and scattering the disease elsewhere, thus shortening the journey of the patient to the grave. Looking back through that night of meddlesome, though innocent surgical transgression, as I view it in the long experience of after years, and fully mindful of the fact that the universal sentiment of authority counsels immediate surgical removal of all growths in the larynx of the tubercular subject, I must confess that even now I approach the consideration of their treatment with great trepidation as I give you the advice, which I gave in this college, but before another society, over twelve years ago. The mere presence of a tubercular tumor in the windpipe is not always necessarily an indication for its removal. If an operation is to be done, it should be done for good and sufficient reasons, and after weighing carefully all the facts in each individual case. Tubercular tumors of the larynx, as far as we at present know, pursue a slow course, show little tendency to early ulceration and may survive with unbroken surface the process in the lung. In their removal by ordinary methods the possible dangers of autoinfection, with metastasis and reinfection at the seat of operation, should not be lost sight of. That these dangers are not chimerical is apparent from some of the recorded literature of the subject, and notably in the case of Hennig, in which death from reinfection took place thirty-seven days after the operation. In my own cases two were found postmortem, two were already past all surgery, while the fifth remained with unbroken surface for ten years, and was not touched with instruments of any kind. Serious interference with function should, of course, constitute ground for operation, and the character of the latter will depend on the nature of the case. Whatever method of procedure is adopted, it must be radical and include not only the removal of the growth in its entirety, but also a liberal portion of the surrounding healthy tissue. A closer study of tubercular tumors of the larynx will be necessary before we can formulate a definite plan of surgical treatment. In the meantime, we must watch and wait.

Out of all the joyous hours at Golden Square that still linger affectionately in my heart and in my recollection, there comes to me but one disturbing and discordant memory—our treatment of cancer of the larynx. This consisted, I shudder in the telling, in the performance of tracheotomy, and the subsequent removal, piecemeal, of the growth through the natural passages. By this process, which today in enlightened surgical communities would be considered as a means of slow murder, the growth was stimulated at once into much greater activity; the patient naturally became worse and worse, and was sent to his long home much earlier than if he had been left severely alone. Of course, there was never a thought of cure. The patient was passed around from surgeon to assistant, and from assistant to student, each in his turn removing fragments from the larynx. One of the visitors to the clinic said, on one occasion, of a case thus maltreated, "Look at the poor devil; he has been plucked at by every expert and tyro in the place!" Naturally, the more the forceps were used, the more desperate bcame the plight of the patient. These were in very truth the days of frightfulness in the management of this disease. As I have often said since then, when I look back through the years in which I have seen cancer of the larynx maltreated, and in which I have unconsciously maltreated it myself, I am simply appalled at the retrospection. With the accusing voice of those days of sacrifice and slaughter still ringing through my mind, and in full view of the chastening experience of succeeding years, I trust that in the long and bitter fight that has raged around the treatment of cancer of the upper tract during the two last decades I have at least in part atoned for the sin of my youthful experience, although I have been held up as a still greater transgressor for demanding in all cases of larynx cancer the most radical measures, and for keeping hands off the growth until the last. Just here permit me to correct a wrong impression that seems to have been created in the minds of some of my colleagues, both at home and abroad, as to my views on microscopic evidence in the diagnosis of suspicious looking neoplasms of the larynx. According to my critics, I reject completely the use of the microscope in the diagnosis of malignant growths of the larynx, and therefore would recommend the complete operation for that disease in the presence of doubt as to its nature. As one of them puts it, I "kick the microscope into the dust heap." No one but a congenital fool would refuse in doubtful cases the aid of the microscope, and no one outside of an asylum would advise a radical operation (such as the one suggested by me) without a certainty of diagnosis. There are some things that go without saying and which ought to be obvious to the dullest apprehension, and I cannot think that anyone who knows me can believe me guilty of such insanity. My original remarks made in 1900, which have called forth such a storm of abuse and misrepresentation, dealt in general principles of diagnosis, and no attempt at elaboration or specification was made. My position, as then stated, is simply that the microscope should be the court of last resort—the final method of appeal. Hands off the growth until the last. Then, if microscopic examination is necessary, let patient and surgeon be prepared for immediate operation. As I said on the occasion already referred to, "before resorting to thyrotomy as a diagnostic means in general, especially if a portion of the growth is to be removed for examination, it should be clearly understood beforehand with the patient that, if the disease should prove to be cancerous, the surgeon shall be at liberty, if in his judgment it seems best, to proceed at once to operation." I took this stand in order to check, if possible, the reckless and indiscriminate removal by laryngologists of suspected tumors of the larynx for microscopic examination, and from what I hear and read I may be pardoned if I say that the warning has not been given in vain.

As these remarks are devoted to reminiscence and confession, and not to controversy, a discussion now of the subject would be entirely out of place. As I leave it, let me turn to the last words on larynx cancer by Butlin, the inspiration and leader of the English school, uttered not long before he died. "I wish I had begun to perform it (laryngectomy) earlier. I am sure that several of the cases in which I performed thyrotomy were much better fitted for laryngectomy, and I cannot help thinking I might have saved one or two of the patients in whom recurrence took place if I had then removed the larynx. I think the glands ought to be removed in every case in which there is extensive carcinoma of the larynx, even if it be intrinsic, unless the disease is limited to the middle zone of the interior of the larynx. Even in these cases it would probably be a wise precaution to remove the glands. I have never removed the glands and the larynx at one sitting."

Catarrhal affections of the larynx were treated with inhalation, insufflation, and topical applications were made to that organ with the camel's hair brush, the use of compressed air being then unknown in England. Our main reliance was on the salts of zinc, the chlorid and the sulphat in simple, and the sulphat of copper in syphilitic laryngitis, and I may say in passing that there are few, if any, agents that surpass in efficacy in simple inflammation of the larynx these preparations of zinc, which are among our oldest and most trustworthy servants in the treatment of this class of affection. Among the ward formulæ in Bellevue Hospital was one which we greatly relied upon in the treatment of venereal warts, and which consisted of a solution of sulphat of zinc in spirits of lavender. When this was applied to the growths, they vanished as if by magic. So striking was the astringent effect that when I started in practice I had a more elegant preparation of the zinc salt made, which I have been using successfully every since.

Comparatively little was known at that time of affections of the thyroid gland. All cases were alike to us—all were goiter. The treatment consisted either in drawing a seton through the tumor or embedding in its substance a dart of zinc shaped like the point of an Indian arrow and inserted into the body of the growth through a liberal opening with the knife. Profuse suppuration was thus set up, which after a lapse of more or less time, and after much inconvenience and suffering, caused, or did not cause, diminution in size or virtual disappearance of the growth. A photographic album was kept in the clinic with lifelike pictures, which preserved the images of the patients in the various stages of their martyrdom.

Turning now to diseases of the nose (rhinology), we find that, with the exception of polypus and ozena, it was practically a closed book. Even with deflection of the septum (can you think it?) we had no concern. The same was true of the accessory sinuses. In the second volume of Morell Mackenzie's classical work, published as late as 1884, in the section on nasal diseases, the subject is not even mentioned. Even the antrum was overlooked, in spite of the fact that centuries before, Drake and Cowper, and the two Meibomii (father and son), had lent their influence to the necessity of the investigation of antral diseases, and to the surgical methods for their relief. And Palfyn, in the seventeenth century, had called attention to frontal sinus suppuration, and proposed the trephine for its cure. All that I knew about the sinuses was contained in an article by Sir William Hamilton in the *Medical Times,* 1845, and familiar to me in my college days through its reproduction in an appendix to his well-known work on metaphysics. In this curious contribution he sought to combat the dogmas of phrenology by showing, among other things, that the frontal sinuses were the natural abodes or hiding places of many different kinds of worms and other low forms of life.

"The motley multitude of its guests might almost tempt us to regard it as

"The cistern for all creeping things
To knot and gender in."

Much confusion existed in those days of the sixteenth and seventeenth centuries as to the source of purulent discharges from the nose, some considering them all of cerebral origin, others declaring that those which were not attended with fever, headache or pain elsewhere, and from which the pus flowed from the nostrils without inconvenience to health, as in a suppurating ear, came from what was known then as the pituitary sinuses. Nathanael Highmore, who wrote at that time, and whose name has been preserved from oblivion by his graphic account of the antrum maxillare, did not help the existing confusion much when he declared that the ostium maxillare which he described and depicted, was an immisary, and not an emmisary, foramen of that cavity. Hence those who followed him described pain in the teeth and caries as due to a "humor distilling from the head into the antrum of 'Highmore,'" thus getting the cart well in front of the horse. Let me remind you here that in the ancient Greek systems of medicine, all nasal discharges, whether catarrhal or suppurative, were supposed to come from the brain, through the cribriform plate, ethmoidal and sphenoidal cells, according to Hyppocrates; from the pituitary gland and ventricles, according to Galen. These views of the Greek physicians, whose notions of the etiology of disease were curiously influenced by the prevailing philosophic doctrines and vagaries of their time, were followed by the Arabian school, and were imported by them into Europe, and prevailed on the Continent as late as the seventeenth century, when they were completely overthrown by the colossal labors of Conrad Victor Schneider, whose wonderful anatomic picture of the nasal mucosa led Haller to christen it the Schneiderian membrane. It is true that van Helmont had long before assailed with pitiless satire the "gray-haired dreams of the Grecians"; that Cardan had previously shown that the discharge came sometimes from the secreting portions of the nasal membrane, and that Botal had entered an anatomic protest against the hypotheses of the ancients, but it is chiefly due to the exhaustive anatomic researches of Schneider that their absurdity was demonstrated. Schneider's demonstrations imparted a great impetus to the study of the anatomy and surgery of the head. Morgagni laughed at Highmore's blunder, but conspicuous among the surgeon anatomists of his day, it was William Cowper, who was the pioneer, and who shed most light on its differential diagnosis and treatment, and made sinus suppuration give up the secret which it had kept so long.

But in spite of the fact that the subject had a literature running back to remote or even ancient times, it was not until the great grippe epidemic in the declining days of the century that has recently passed away that man first awoke to the full realization of what a curse his sinuses had been to him (and he, too, all unconscious of the fact) throughout all the centuries that had gone before. At that time, while we had made great advances in technic, while the dawn of a new era in this class of affection was breaking, it must nevertheless be confessed that we followed very closely the teachings of our masters of long ago.

In 1894, at the Congress of American Physicians and Surgeons, the subject of accessory sinus disease was given prominent place, for the first time in this or other countries, before a general medical and surgical audience of the entire society. Bosworth, Bryan and I were assigned to the task of opening the debate. The following year the first international discussion of the subject of the surgery of the sinuses was held in London at a special and unusual session of the British Laryngological, Rhinological and Otological Association. Those who took part in it and gave their views and experiences were Bosworth, Luc Delavan, Moure, Lennox Browne, Daly, Mayo Collier, DeRoaldes, Dundas Grant, Krause, Bryan, Bark, Sajous, McIntyre and Stoker. For some inscrutable reason I was asked to open the debate. The remarks of those who followed me were most instructive, and for that day a fairly thorough exposition of the then status of the question. I mention these two occasions, for they were among the early signals for the onrush of the coming events in this special department of surgery, which had already cast their shadows before. The ball had started to roll, the pot had commenced to boil; the worst was yet to come.

No, I shall not disturb the tonsil question. God forbid! I am down in cold, remorseless type in many places on that subject, and even if I wished to run away from my convictions I would have no avenue of escape. I only want to say that those far-off days at Golden Square were perhaps the tonsils' happiest and most halcyon time; for it had not then been found out that the germs of a multitude of diseases common to man left well-established, convenient and natural avenues of entrance into the body to seek their destination by other and more devious paths through the tonsil substance; nor had this invading host of pathogenic visitors, some well known, others nameless and nondescript, yet found in the tonsil crypts and in the tonsil vessels either a birthplace or an asylum. They fell; yes, fell as leaves in Vallombrosa. Not a day passed that did not take its bloody toll of tonsils. The guillotine was nearly every day much the busiest instrument on the job. In all that long roll of cases I fail to recall a single serious accident after tonsillotomy, except now and then severe hemorrhage; nor have I ever seen since then the operation done with more dexterity and thoroughness. If the surgeons of those days did not do as much far-reaching good as the tonsillectomist of the present time, they certainly did infinitely less harm.

One of the strangest things in the early development of rhinology in England and America was the slow and belated perception of the importance and significance of the condition falsely known as adenoids. Although over ten years had elapsed since the publication of Meyer's work on the subject, and although two years later it had been translated into English in the transactions of the Medical and Chirurgical Society of London (1870), very little attention was given to it in the clinic. Woakes and I operated on a great many cases. Woakes wrote a paper in which he maintained the papillary nature of the growths against the generally accepted belief that they were glandular or adenoid in structure. We never knew our mistake until French had to tell us that they were neither papillomatous nor adenoid, but lymphoid in character. Woakes was an interesting personality, with original, but often erroneous, theories. His work on "Necrosing Ethmoiditis," while pathologically hopelessly wrong, served to first rivet attention on the study of ethmoiditis, and was therefore historically the beginning of the modern literature of that affection.

In looking back to the old days in London, allow me to recall yet another experience, which to me at least has a certain historic interest. One morning I received a visit from a friend and fellow-lodger in the house in which I had, as the English say, "chambers." He was a phlegmatic Dutchman, a born linguist (spoke seven or eight foreign languages fluently), a globe trotter, an observer of a most inquiring and eager mind. He had come to consult me about a coryza which invariably and only occurred after sexual indulgence. I told him that he must be dreaming, or that he must catch cold during the sudden cooling off process following the heat of tempestuous bodily exertion. Not at all. He did not get unduly excited at the time. On the contrary, he was more or less indifferent to coitus, but practiced it one day a month as a physiologic duty (or purge), as a matter, as it were, of personal hygiene. It was a part of his philosophy. One night during the month, coitus, the following morning always coryza.

The situation was unique, interesting, absolutely new to me and others to whom I told the tale. Shortly after this experience I stumbled quite by accident in the clinic on two women who complained of stoppage of the nose, sneezing and watery discharge, occurring only during the menstrual period. Not to delay you with a longer recitation of the circumstances in the case, without a guide, in an absolutely unknown territory, coming across an observation here and there and ever on the alert and looking for cases bearing on the subject myself, at the end of five years I had accumulated enough material from which to generalize and publish my conclusions, which I did in the *American Journal of Medical Sciences* for April, 1884, in an essay which was the first attempt to reduce this curious relationship to a scientific basis. This, then, is the simple story of the almost accidental discovery of a then unknown physiologic relationship which today has an enormous literature. Two years ago I received from Germany a work by the younger Seifert, which consisted of a critical review of nearly three hundred brochures, theses and papers on the subject, and even this list of contributions was incomplete.

From London I went to Munich, where I became an assistant in the clinics of Zeimssen and Oertel. Zeimssen was a prolific writer, but is chiefly known to laryngologists as the author of the articles on diseases of the larynx in his well-known Eycyclopaedia of Medicine. Oertel wrote little or nothing on laryngology, but his articles on the physics of laryngoscopy were the most scientific of his day. Zeimssen, a tall, distinguished patrician type of man of advanced years; Oertel, a little hunchback dwarf of middle age, but whose face was intelligence itself, and whose eye was as piercing as the Roentgen ray. Both masters of internal medicine, both experts in laryngology.

Although a comparatively small town, the wealth of clinical material in Munich was enormous. I have never seen in a given period of time, not even in the vast clinics of London, such a number of laryngeal growths, nor have I ever seen them removed with greater skill. They came to Oertel from all parts of the Continent, and the little man, even in the most difficult cases, always made good. For the student the atmosphere was ideal, the combination of special and general work perfect. At one moment we were giving cold baths or packs to typhoid patients, in the next removing a laryngeal growth. The special branch of medicine was not studied apart, but kept in contact and closest touch with all the other departments of medicine. We could not get away from any one part of general medicine even if we wished to do so, and this leads me to say that the proper time to lay the foundation for the educated specialist is in early life, in his early studies, if possible in his undergraduate days, for it is at this period of his medical training that he is in the best position to acquire that fundamental knowledge, and, what is of more importance, that breadth of mind which is so essential to his future development. At this receptive period of the development of his mind he can best recognize the limitations of each special branch of medicine, and can best be taught to generalize profoundly not in one, but in all departments of medical thought It is at this stage of his career that it is impossible for him to cut loose from the other departments of medicine. The growth of his special studies goes on pari passu with his advance in other lines of work, and he is brought in daily contact with disease in other organs of the body. He learns at the outset that no department of medicine is isolated and independent, but that they are all mutually dependent and coordinate. And if he is made of the proper stuff, this fundamental lesson and illuminating first impression will follow him in his special work in after life as an inspiration and a guiding star.

Those days in Munich were halcyon days. I thank my stars that I lived and learned in the older Germany, before that nation had altogether ceased to be the dreamer that she was when she entered the modern world; that I heard her music and listened to her songs in the joyous, peaceful days of the Lorelei, and not in those of the Hymn of Hate; before the departure from simplicity and simple idealism of the lovable people who gave the world Santa Claus.

Later, when I studied in Vienna, I found a different atmosphere. Here the only means of acquiring special knowledge of the subject were the imperfect courses on diseases of the larynx given by the professors and their assistants. There was no special course in rhinology, which subject, as in England, was left entirely alone. Stoerck and Schroetter were the leaders in laryngology at that time. The former, although handicapped somewhat by his method of examination, the socalled Schusterkugel, and a trembling hand in manipulation within the larynx, always delivered the goods. Hans Chiari, then assistant in pathology, now professor in Strassburg, had charge of the department of pathology in the Rudolfspital. He had five young men to help him in the laboratory, three of whom were Americans—Councilman, now of Harvard; Belfield of Chicago, and myself. It was one of my duties at the autopsies to look after the condition of the larynx and trachea, so that when one morning the body of a man who had died of cancer of the stomach was brought in for a postmortem, I was handed the windpipe for examination. Secondary cancerous deposits were present in the liver, kidney, spleen and other organs. The lungs, however, contained tubercular cavities; the pharynx, larynx and trachea were free from inflammation and ulceration. The bronchial and retrotracheal glands were enlarged, tumefied and caseous. In the membranous posterior wall, at its junction with the cartilaginous framework of the trachea, about one and one-half centimeters above the bifurcation, was a well-defined circumscribed tumor about the size of a small bean, its long axis parallel with that of the trachea, and of a uniformly even, smooth appearance. It was covered by the mucous membrane of the trachea, and was dense in consistence, giving to the touch the sensation of a hard cancerous nodule, for which, indeed, it was mistaken. A similar growth was found in the pericardium. The microscope revealed a picture for which I was not prepared. It showed, namely, that the tumor, which seemed to have its origin in the submucous connective tissue, consisted in the main of an aggregation of distinct tubercular nodules, set in a more or less well-marked vascular network of connective tissue. The majority of the tubercles lay in the deeper portions of the mucous membrane and in the submucous tissue. A few were more superficial, lying under the epithelium. They exhibited all grades of degenerative change; in some caseation was so far advanced that nothing remained but the cellular wall. Between the individual nodules the connective tissue was hypertrophied and the seat of a moderate amount of round-celled infiltration, which had invaded the glandular follicles in its vicinity. The tissue of the trachea in the immediate neighborhood of the growth presented no remarkable change. The nodule in the pericardium showed the same histologic structure that was found in the tracheal neoplasm. Shortly after this experience I came across a similar case, in which the tumor was confined in a most unusual way to the vestibule of the larynx. The subject from whom the growth was taken died of pulmonary tuberculosis. The whole upper compartment of the larynx, including the epiglottis, aryepiglottic folds and ventricular bands, presented a remarkable appearance. It was completely covered by little mounds, which represented small, uniformly smooth, dense, moderately hard nodular growths, which lay beneath the mucous membrane. The nodules were about the size of a split pea, each merging into its neighbors, so as to form one continuous growth. The process ceased abruptly on either side at the free border of the ventricular band. There was no trace of ulceration in pharynx, larynx or trachea. Microscopic examination of numerous sections of the growth showed it to be of the same nature as the above-described neoplasm in the trachea. These two cases were absolutely unique. Chiari had never seen anything like it before; no one had even suspected the existence of such a condition. After over a year's search through literature for similar cases, I published my own in the *Archives of Medicine*, New York, for October 1, 1882. As much confusion still exists as to what constitutes a true tubercular tumor of the windpipe, I may be pardoned for again drawing attention to the subject. Since the publication of my own, cases have here and there found their way into medical literature, some without doubt examples of true tubercular tumor as defined in my original article, whilst others—and these are probably in the majority—are extremely doubtful in nature, and must be thrown into the category of localized infiltration or into the papillomatous group, which I have already considered. A true tubercular tumor is extremely rare, and by true tubercular tumor I mean not simply any localized swelling containing the bacillus, but a distinct, definite characteristic tumor formation covered by unbroken epithelium and consisting of a congeries of miliary tubercles set in a vascular network of connective tissue and exhibiting all grades of tubercular degeneration to cavity formation. I have seen but three cases in which the diagnosis was microscopically established beyond a doubt, and two in which no histologic examination was made. As far as my limited experience goes, the tendency of this form of tumor as well as the papillomatous variety is not toward ulceration unless tampered with by incautious attempts at instrumental removal. In one case, in which the patient made a gallant fight for life of nearly ten years and finally succumbed to pulmonary hemorrhage, the growth, which consisted of a small, smooth lobular tumor in the interarytenoid fold, during that whole period, beyond a slight increase in size, remained practically unchanged. These two cases are of historic interest, inasmuch as they are the first cases on record of tumors of any kind in the windpipe shown microscopically to be tubercular. They therefore represent the earliest exact knowledge of this form of tuberculosis, and are the first to establish the separate existence of this previously unknown phase of that disease.

As in London, so in Munich and Vienna, no attention was paid to the nasal passages or accessory sinuses, and therefore no courses on the subject were given. I had to turn my steps homeward to learn something about the diseases of these organs. Here, too, I entered a practically untrodden field. The subject holding the center of the stage and overshadowing every other at that time was the surgery of the turbinated bodies, and especially the inferior, in the treatment of the hypertrophic form of rhinitis, and the operation engaging most attention was the removal with the cold wire snare of the posterior hypertrophied end of the inferior body for the relief of that condition. Although these masses were probably removed again and again with the wire, as anyone can convince himself by referring to the standard surgical works of the last two centuries, their true antomic significance and relation to nasal inflammation was not properly appreciated until Bigelow demonstrated the erectility of the tissues concerned in their development. As I have pointed out elsewhere,[2] Bigelow was not the first to show the erectility of that structure, but to him, apart from independent discovery, belongs the credit not only of giving the best description of this tissue, and of more accurately defining its minute structure and extent of distribution, but also of showing that the so-called mucoperiosteum of the posterior part of the septum is in reality an erectile substance. Bigelow was also the first to observe the alternate inflation and collapse of these bodies, which he compared to that of the lungs of a small animal, thereby leading the way to the rational interpretation of nasal inflammation. From their resemblance to the cavernous bodies of the penis, Bigelow gave them the name of turbinated corpora cavernosa, but as Henle and, more recently, Zuckerkandl have pointed out, they may be with more propriety classed among the contractile as contradistinguished from the erectile tissues.

The credit of urging the necessity of their removal by the cold wire snare belongs to Jarvis. Prior to that the galvanocautery had been used in this country and Germany. In the latter country it was extensively adopted, and it is a noteworthy fact that Zaufal, who is an enthusiastic advocate of the cold wire snare in the removal of polypi, recommends the cautery for turbinated hypertrophies. .Jarvis was followed by Bosworth, who wrote enthusiastically upon the subject. I wrote about it, everybody wrote about it, and everybody operated. One of my colleagues said that, on leaving my office one day, he trod on what he thought was a lot of peanut shells, but on picking some of them up he found that they were turbinated bones. The operation in the course of time was abandoned. The operators went to the other extreme, as in the case of all surgical crazes. The operation, however, within proper limits did much good, for the following reasons: In cases suitable for it, it fulfilled in a simple and radical manner the removal of the obstruction, and it did this without interfering with the normal air currents in their curvilinear course through the middle meatus, as that avenue and the anterior portions of the passages remained unchanged in their anatomic relation, thus preserving intact normal respiration and natural filtration of the air. As the hypertrophied mass meant tissue largely deprived of function from the loss of glands and blood vessels, their absence involved no sacrifice of physiologic usefulness, while their removal not only facilitated cleansing of the posterior nares, but also diminished apparently the amount of tenacious mucus which they contributed to the nasal discharge. It also seemed to relieve the collateral congestion in other parts of the cavernous structure. Finally, in some cases it caused the complications of socalled reflex character to disappear, especially cough and asthmatic breathing. The latter is easily understood when we reflect that it is this portion of the reflex sensitive area of the nose that is most responsive to reflex producing impressions, notably those concerned in the production of cough and asthma.*

The simple removal of the posterior end, leaving the rest of the structure intact, is a safer and much more rational procedure than the wholesale destruction of the turbinated bodies, which is the routine practice so common at the present day of those who forget that the aim of nasal therapeutics is preservation of function, and not the destruction of everything in sight. Let me repeat in this connection what I have said upon another occasion:

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*The effects or sensations produced by irritation of the nasal mucosa, whether by mechanical, simple tactile or electric contact, chemical or thermic agencies, or by a pathologic process, are more correctly appreciated and located in the anterior than in the posterior portions of the nasal passages. The more we recede into the deeper regions, the nearer we approach the pharynx, the more vague and indeterminate and inexact are the messages which stimuli carry to the central nervous apparatus. Thus, for example, in stimulation of the lower and posterior portions of the nasal cavity the sensation of irritation or hurt is, in many cases, referred not to the nose itself, but to the larynx, and in some instances to the bronchial tubes, producing in the one case cough and in the other bronchial wheezing. This is not only experimentally but clinically true. Now in calling attention many years ago to a special sensitiveness to certain reflex producing impressions in the nasal mucous membrane, I did not, nor do I now, as has been wrongly inferred, desire to maintain that pathologic reflexes may not originate from other portions of the

No one questions the frequent necessity of the complete or incomplete sacrifice of the middle turbinate bone, notably in the case of accessory sinus suppuration and the radical cure of tumors of the nasal and accessory cavities, and, in occasional cases, as a substitute for operation on the septum. But to make it responsible for a host of woes unnumbered, and to attack it surgically from a purely theoretic standpoint, is vicious both in principle and practice. Especially preposterous is its removal for the alleged cure of the disease falsely called hay fever. In dealing with this structure, it should always be remembered that its anterior end is one of the chief buttresses against the admission of foreign matter to the air passages, the principal point at which filtration of the external air takes place. It should, therefore, not be assailed on indifferent and insufficient pretext, or sacrificed on the altar of fantastic hypothesis.

One of the most interesting, if at the same time one of the saddest, chapters in the books of rhinology is that which tells the story of the surgery of the septum. In the early days of my special practice, comparatively little had been done to rad-

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nose, for wherever there is a sensitive nerve filament it is possible to provoke a reflex movement.

My contention is simply this: that the area indicated in my original paper represents by far the most sensitive portion of the nasal cavities, and that pathologic reflex phenomena are in the large majority of cases related to diseased conditions of some portion of this sensitive area. That all pathologic nasal reflexes arise from irritation of this particular area is a preposterous proposition which I do not and never have maintained. Whether a special sensitiveness in certain portions of the nasal mucous membrane exists or not, the agitation of the question has led to more rational methods of procedure in the treatment of a large class of nasal affections and to more conservative methods in intranasal surgery. Before the location of the sensitive area or areas, the nasal tissues were destroyed with an almost ruthless recklessness that bade fair to bring intranasal surgery into the worst repute. (For an elaborate discussion of this whole subject, see article by the author in Wood's Reference Handbook of the Medical Sciences, edited by Buck, Wm. Wood & Co., New York, 1887, Vol. 5, pp. 222-242.)

ically and satisfactorily remedy abnormalities of that structure; observers busied their brains with the burning question whether the deflected septum is turned more frequently to the right than to the left, never concerning their faculties with the remedial aspect of the subject, and in the presence of that overshadowing conundrum the treatment of the condition was lost, while many followed the ancient advice of Marcus Aurelius Severinus, who declared that, inasmuch as the distorted system was doubtless placed in that position by the will of God, it would be eminently sacrilegious to interfere with such conspicuous manifestation of Divine Dispensation, and therefore it should be left severely alone.

The measures then in vogue were the Blandin artificial perforation, a mischievous and lazy performance; the Adams' operation, the forcible restoration of the septum to the middle line with forceps, a very unsatisfactory and clumsy undertaking: stellate fracture, commonly known as Steele's, and later as the Asch operation, and the saw. Let me stop right here to do a dead man historic justice. The principle involved in the stellate fracture originated with James Bolton of Richmond, Virginia, who many years before described his method of procedure in the *Virginia Medical Monthly*. Bolton first employed an ordinary pair of buttonhole scissors, with which he made the stellate incisions. Subsequently an instrument was made for him by Tiemann of New York, and from this developed the apparatus used at the present day.

Later the introduction of the surgical drill, first driven by the dental engine and afterwards by electric power, marked a decided advance in surgical procedure. With the arrival of other and newer methods this excellent agent fell into undeserved neglect, in spite of the fact that a variety of work, and delicate work, can be successfully done with it without interruption of the duties of the individual, and without disagreeable after-results.

But in spite of these improvements in the surgery of the septum there were cases which still baffled the most ingenious methods. This was particularly true in regard to irregularities in the conformation of its bony framework, as in certain cases of deep-seated deflection in which the patient would not submit to operation or the surgeon hesitated to perform it. To overcome this difficulty I proposed in 1882, at the annual meeting of the Virginia State Society, and carried out in the spring of the following year, 1883, an operation which consisted essentially in the removal of portions of the external wall, and notably the inferior turbinated bone of the obstructed side (or portions thereof) as a substitute, in suitable cases, for operation upon the septum itself.[3]

The method is applicable in cases in which sufficient reason exists for not operating on the septum. Its principle may also be extended in its application to other surgical procedures within the nasal passages. Its chief value is in certain exceptional cases in which other methods are contraindicated or can be carried out only with difficulty.

Since my original article was published I have received several communications from various laryngologists in this country, notably from the late Jarvis, and Roe, of New York, in which they have stated that the principle had not only given them great satisfaction, but had also extricated them on more than one occasion from great difficulty and embarrassment.

The crying need in operation on the septum is greater simplicity of performance. With a comparatively speaking small armamentarium, backed by a quantum sufficiat of good, common, surgical horse sense, the surgery of the septum is not a complicated problem, but one which can be worked out with comparative simplicity and satisfaction.

Such was the state of the question thirty-three years ago. You know its subsequent history. I will not, therefore, carry you any farther into paths which are perfectly familiar to you, and will leave you now with this brief review of the older methods, bidding you remember that it is a long, long way from buttonhole scissors to the complicated technic of submucous resection, and asking you to graciously accept this little bit of personal experience and reminder of what we had to contend with in the earlier days from one who has traveled through the dust and heat of that road.

At a banquet held several years ago by the American Laryngological Association, the toastmaster, Kelly Simpson, in calling on Bosworth for a speech, said that his (Bosworth's) saw would be still on deck long after the swivel knife had been forgotten. While this may sound to some as the irresponsible language of postprandial enthusiasm and exaggeration, at the same time it may well come to pass that in some cases we will return to some of the older methods, or modifications of older methods, which, though less spectacular than most of those performed at the present day, did very much less harm.

The elder Disraeli, in the preface of his "Amenities of Literature," says that to be ignorant of all antiquity is a mutilation of the human mind. It has long been my custom when seeking something new to go to the ancients. So in the rush of modern invention and procedure it may sometimes be wise and profitable to pause, retrace our steps, and pass in review the work of those who have gone before. We of this generation may in this way learn much from those who, in the gray dawn of its earlier history, fought the battles of laryngology to make its calling and election sure in the eyes of their fellow-men.

In this rambling, disorderly, crazy quilt talk which I have given this little family gathering here assembled, I have endeavored in an informal way to recall some of that past, to remind you that all things did not go very well then; that difficulty after difficulty had to be met and surmounted; that the distrust in which our calling was held in many quarters had to be overcome, and that finally the laryngoscope had to be brought from its lowly place as a simple means of examination to occupy its now recognized lofty position as an instrument of progress and power in medical research, in scientific expansion and in the exploration of the unknown. The men of those days lived in a time of crisis. Laryngology was on trial, its fate was hanging in the balance. An obsession or madness such as some that have since swept through the specialty would have turned the scales, for the serpent of Esculapius had not then been driven from the temple by the Golden Calf. Remember, too, that, in that day, the earnest seeker after truth had to reckon not only with his brother in regular standing, but also with the charlatan and the professional quack. It was this latter gentry's quickening opportunity and appointed time; for in the darkness and mystery of an incompletely explored terrain they found their Canaan, and in that promised land their fields of gold. Medical men confounded them with the struggling laryngologist, whom they looked upon with suspicion, and spoke contemptuously of his limited sphere of work, quite forgetful of the fact that their own ignorance of the subject was abysmal, that their sole weapon for the conquest of throat disease was the probang, and that they were long unwilling to let even that frail scepter of their power, or impotence, pass to other hands.

I have tried this evening to live only in the past; to forget the present with all its wealth of marvelous achievement; to call back a part of the practice of a rugged era, some of which deserves to outsleep Endymion in oblivion, but much of which was most wholesome and good; to breathe again the purer and the happier atmosphere of that era, and to snatch from it, perhaps, some lost ideal; to roam once more through pleasant fields, wild and uncultivated maybe, but which loving recollection has forever kept full of fragrance, even though the budding hope of laryngology had not then come to flower.

ADDENDUM.

The equipment of the medical student is not complete without some knowledge of the past history of his art and the names, the personalities, the aspirations, the ideals, the lives and deeds of those who made it. It is an amputation of his mind to cut it off from the wondrous story of the birth and growth and development of medicine and its compelling progress in all the ages, through storm and sunshine, through error and enlightenment, through failure and achievement. And if he goes at it in the proper way, its study will not be labor, but will bring to him the most satisfying mental relaxation and the most refreshing consciousness of culture that cannot be obtained in any other way. In the short sketches which follow I have given a few brief references to the work of some of the men whom every student of rhinology should know something about.

Jean Fernel, or Fernelius, as he is known to the medical historian, was born in Picardie (1496), but later moved to Paris, where he acquired a large practice and became body physician to Henry II, then King of France, whose sterile wife, the notorious Catherine de Medici, was made to bear offspring through his skillful treatment, a service which so pleased the monarch that whenever he left home he always took Fernelius with him. On one of these excursions the physician's wife was taken suddenly ill with a cold and died, and her husband's grief was such that he followed her soon afterwards to the grave. Catherine had a number of children, among them two daughters—Elizabeth, who married Philip II of Spain, and Margaret of Valois, who married the King of Navarre—and a son, Henry, who ascended the throne as King Henry III. It is interesting in this connection to recall the anecdote of the latter at the betrothal feast of Margaret, as an historic example of the power of olfactory impression in awakening sexual desire. On that occasion he dried his face, by accident, with a garment moist with her perspiration, belonging to Maria of Cleves. This so excited him sexually that, although Maria was then the bride of the Prince of Condé, he could not restrain his passion, and made her miserable, as history tells us. A similar story is told of Henry IV and the beautiful Gabriel, who later became his mistress, the King's desire being aroused when at a ball he wiped his wet brow with her handkerchief.

Fernelius was a prolific writer, but among his services to medicine he should be remembered by laryngologists in general and students of sinus disease in particular as the keen observer, who a century and more before Schneider wrote, with the ancient doctrines of the Greeks and Arabians still unchallenged, and four hundred years before our own time, drew, unconsciously perhaps, a subtle distinction between suppuration from the "pituitary" sinuses or thereabout and the purulent flux which was supposed to come from the cavity of the cranium.

Gabriel Fallopius (1523-1562), famous anatomist, whose name survives in many parts of the body, notably the Fallopian tubes, the brilliant pupil and successor of Vesalius, should be familiar to laryngologists for having introduced the cannula in the development of the modern snare and as the alleged discoverer of the sphenoidal sinus.

Aranzi, Arantio, or Julius Caesar Arantius, a celebrated Italian physician and surgeon of the sixteenth century, wrote, among other volumes, a work on the human fetus and tumors, and invented a forceps for the removal of nasal polypi. He introduced an original method for the direct examination of the nasal passages. In a wooden window shutter, opening into a darkened room, he cut a circular foramen, through which the direct concentrated rays of the sun were allowed to stream. The organ to be examined (nose, ear) was thus illumined by the column of sunlight. If the sun was not shining, or if the examination took place after dark, he used a candle or, better still, a globe of crystal filled with water through which the light was made to pass, thus anticipating the socalled "Schusterkugel" method of examination of the present day by nearly three hundred years.

Nathanael Highmore, an English anatomist, born in 1613, the author of books on the art of generation, hysteria and hypochondriasis, is chiefly known to medical scholars by his work on anatomy, published in 1651 and entitled "Corporis Humani Disquisitio Anatomica," in which occurs his famous description of the antrum maxillare illustrated by a large plate with three or four figures, in which he portrays the cavity and its relation to the surrounding structures and to the brain.

The two Meibomii—John Henry and Henry, his son, the latter born in 1638 and known to anatomy as the discoverer of the Meibomian follicles in the eyelids—were among the early investigators of antrum disease, as we learn from the work of J. G. Günz (Programma Indicit et Observationem Ad Ozaenam Maxillarem Ac Dentium Ulcus Pertinentem Proponit —Lipsiae, 1753), a learned exposition of the maxillary antrum surgery of his day.

James Drake (1667-1707), an English anatomist and surgeon, chiefly remembered by his classic work on anatomy entitled "Anthropologia Anatomica," was one of the first, if not the first, to call attention to the fact that the sanies of ozena (fetid and purulent discharges from the nose) came not infrequently from the pituitary (accessory) sinuses and more especially from the antrum.

William Cowper, an English surgeon anatomist, born in 1666, has the credit of having discovered the urethral glands (1702), which today bear his name, although they were previously found and described by Méry (1684), wrote several well-known works on the muscular system and a treatise on anatomy entitled "The Anatomy of Humane Bodies," published in 1698, which he illustrated with one hundred and five original plates, to which he added nine perfunctory ones of his own, which, with a serene audacity which is not a stranger to some of our fellow-mortals of the present day, he boldly stole from the classic work of Bidloo (1685), a celebrated Dutch anatomist and contemporary. Cowper's service to laryngology consisted in bringing the main anatomic facts of sinus (antral) disease to light, in laying open, as it were, its pathologic condition, in showing how it could be distinguished from other affections, how easy it was with the aid of anatomy to apply remedies direct to the cavities themselves, and finally how readily the structures could be reached surgically, thus making a giant stride from the practice, which we learn from Celsus, came down from a remote antiquity, of cutting open the face even in cases of superficially situated ozena and purulent discharge and sewing it up again.

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III.

THE ETIOLOGY AND PATHOLOGY OF OTITIC CEREBELLAR ABSCESS.

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Although recent otologic literature contains many case reports of cerebellar abscess, there are but few carefully worked out pathologic examinations of this interesting condition.

Cerebellar abscess may complicate either an acute or a chronic suppuration of the middle ear and mastoid. It is, however, far more frequently associated with the chronic otitides. In the eighty-six cases which we have been able to collect from the literature since 1906 (the date of publication of Bárány's first important work on vestibular nystagmus), fourteen (16.2 per cent) have complicated the acute otitides, while seventy-two (83.8 per cent) have followed the chronic suppurations.

The reason for this undoubtedly lies in the fact that in the chronic cases there is greater destruction of bone, prolonged interference with drainage, and the presence of cholesteatoma. Thus in the vast majority of instances we find either polypi, cholesteatoma or small tympanic perforations, chiefly in Shrapnell's membrane. Furthermore, in chronic suppurative cases there usually occurs a sclerosis of the cortex which favors necrosis of the deeper lying bony structures. It is particularly in those instances in which cholesteatoma has eroded the bone and lies in direct contact with the dura that intracranial complications occur. The cholesteatoma not only seriously interferes with drainage, but also acts as an excellent medium for the growth of the invading microorganisms.

Males are attacked much more frequently than females, the proportion, in our series, being as three to one.

Neumann gives the following statistics on the relative frequency of occurrence of cerebellar abscess at different periods of life:

Author.	0-10 Years.	11-20 Years.	21-30 Years.	Over 30 Years.
Koch	11	33	35	19
Okada	17	56	45	28
Körner	4	14	12	10
Heimann	16	57	38	26
Neumann	12	51	41	30

In the series here reported it occurs with greatest frequency in the second decennium, and with least frequency in the first and sixth decennium, while in the third it occurs about as frequently as it does in the fourth and fifth combined: Thus in the eighty-six cases, six were under ten years; twenty-nine were between ten and twenty years; twenty-two were between twenty and thirty years; twelve were between thirty and forty years; ten were between forty and fifty years, and five between fifty and sixty years. In two the age was not stated. It occurred forty-five times on the right side and thirty-seven times on the left. In four the side was not stated.

With regard to the etiology of cerebellar abscess Neumann differentiates between those which complicate the acute middle ear infections and those occurring with the chronic otitides. In the fourteen cases of cerebellar abscess complicating acute otitis media and mastoiditis, sinus thrombosis occurs in six. In only one, however, is there a direct connection evident macroscopically between the infected sinus and the cerebellar abscess. This proportion is probably too low, and in other statistics such evidence is more frequently met with. Although in the majority of instances the cerebellar infection is secondary to the sinus thrombosis, this does not seem to be invariably so.

Death from the rupture of a cerebellar abscess into the lateral sinus has been reported, and the fact that symptoms of sinus thrombosis occur late in some of the cases, notably those of Barr, Beck and others, brings to mind the possibility of the sinus infection being secondary to the brain abscess. Histologic proof of this etiologic relationship is not at hand. In four of the acute cases an epidural abscess in the posterior fossa was probably the pathway of the endocranial complication. So that in our series, too, the route which the cerebellar infection followed in the majority of cases complicating acute otitis was either from a thrombosed sinus or an epidural abscess. In but one of the acute cases (Uffenorde) was the labyrinth the pathway of the intracranial infection. This was an acute middle ear infection complicating scarlet fever, and the cerebellar abscess resulted from an extension of a saccus empyema. Here, despite the virulence of the infection and the lack of resistance on the part of the individual attacked, the intracranial extension from the suppurative labyrinthitis was a circumscribed collection of pus—i. e., a brain abscess—and not a diffuse purulent meningitis.

From an etiologic point of view the cerebellar abscesses which complicate the chronic middle ear suppurations differ very decidedly from those which occur with the acute otitides. In Neumann's series there are one hundred and thirty-two cases of cerebellar abscess complicating chronic suppurative otitis media. Twenty are not sufficiently studied. Of the remaining one hundred and twelve, forty-nine—that is, 43.75 per cent—showed labyrinthine suppuration. In the series here reported, thirty-one of the seventy-two cases with chronic purulent otitis showed labyrinthine suppuration. These figures, startling as they are, are probably an underestimate rather than an overestimate. We base this judgment upon the following facts: If we examine individual reports included in our series, we find, first, eight cases reported by Michaelsen. Of these, six are cerebellar abscesses complicating chronic suppurative otitis media. In these six cases "dead labyrinth" was found five times. Again, Ruttin reports seven cases, all of them complicating chronic suppurative otitis. In six of the seven, labyrinthine suppuration occurred. Neumann reports five cases, all of them complicating chronic purulent otitis media. In these, labyrinthine suppuration occurred three times. These figures, we believe, are the result of careful clinical examination, and are not accidental. In many of the cases the labyrinthine suppuration was proven histologically.

Undoubtedly there is a small proportion of cases of cerebellar abscess in which, although the labyrinth is the seat of a suppurative process, it is not the pathway of the infection from the middle ear to the endocranial structures. In a case which the writer recently had an opportunity to examine postmortem, and in which the temporal bone was examined histologically, the following facts presented: Not a vestige of labyrinthine end organs, auditory or static, remained. There was a fistula in the external semicircular canal filled with granulations and new formed connective tissue. From this focus the labyrinth probably became infected. The crura of the stapes were destroyed, but the footplate, although much eroded, was still present in the oval window. With the exception of this fistula in the external semicircular canal, the bony canals were everywhere intact. There was, furthermore, no erosion of the inner labyrinthine wall. The internal auditory canal contained no pus. The pathway of the intracranial infection could be traced through the inner (posterior) antral wall. In this situation there was an epidural abscess, and a perforation led through the bony wall and through the dura to the cerebellar abscess.

In our series suppurative labyrinthitis and epidural abscess occurred seven times. Thus suppurative labyrinthitis, epidural abscess and cerebellar abscess form a fairly common triad. The etiologic relationship between these three factors is not a constant one. While it is natural to suppose that the epidural abscess is secondary to the suppurative labyrinthitis, this is not invariably so. In his consideration of the relationship between deep epidural abscess in the posterior fossa and suppurative labyrinthitis, Grünberg reports a most carefully studied case. The labyrinth is the seat of a diffuse suppurative process. The windows, both the oval and round, are intact. The labyrinthine capsule is everywhere intact, with the exception of the common limb—i. e., the junction of the superior with the posterior semicircular canal. At this point there is an erosion from without inward, unquestionably caused by the epidural abscess in this location.

Ruttin has shown that in mastoiditis, particularly in those latent varieties due to infection by the streptococcus mucosus, there is a tendency to necrosis in the deep seated petrosal cells surrounding the labyrinthine capsule. Drainage from this region is particularly difficult, and an epidural abscess may result. It can readily be understood, then, how an epidural abscess in this location may give rise to a cerebellar abscess, on the one hand, and to a diffuse labyrinthine suppuration on the other.

Formerly, in many of these cases of suppurative labyrinthitis, epidural abscess and cerebellar abscess, a saccus empyema was adjudged, upon the macroscopic findings, the pathway of the cerebellar infection. Macroscopic findings, however, are inadequate to establish such a judgment. The microscope alone is the final arbiter.

In eight cases suppurative labyrinthitis occurred with sinus thrombosis. In only one of these, however, is the labyrinth clearly indicated as the direct pathway of the sinus infection. In this instance, in addition to the sigmoid sinus, the bulb, inferior petrosal and cavernous were thrombosed. Here the infection, in all probability, started in the labyrinthine veins, extended to the inferior petrosal sinus, and thence forward to the cavernous and backward to the bulb and sigmoid.

Etiologically, pathologically and clinically, cerebellar abscess may be divided into: (a) Labyrinthogenic, (b) nonlabyrinthogenic.

In all probability at least fifty per cent of cerebellar abscesses complicating chronic suppurative otitis media are labyrinthogenic. In a small proportion of cases the labyrinth is the seat of diffuse suppuration and is destroyed, but is not the pathway of infection to the brain. On the other hand, of those cases of cerebellar abscess which complicate the acute otitides, from ninety to ninety-five per cent are nonlabyrinthogenic—i. e., they take origin either in a sinus thrombosis or an epidural abscess of the posterior fossa.

The pathway from the labyrinth to the cerebellum may follow the internal auditory canal, the aqueductus vestibuli, the aqueductus cochleæ, or a fistula in the bony capsule. In a general way, middle ear infections which progress to the intracranial structures through natural openings, lead to a diffuse suppurative process—i. e., meningitis—whereas, those that do not follow preformed paths, cause circumscribed collections of pus—i. e., either brain or subdural abscesses. The aqueductus vestibuli offers a notable exception to this rule. An infection traveling along this route is retarded probably because of the narrow lumen of the aqueduct, the facility with which protective adhesions are formed, and the fact that the ductus ends in a closed sac between the layers of a very resistant membrane—viz., the dura. Such infections, then, frequently lead to cerebellar abscesses, rarely through a saccus empyema, more often through an erosion of the bony wall of the aqueduct and the formation of an epidural abscess.

Whether the labyrinth infection leads to a diffuse meningitis or to a cerebellar abscess depends upon the virulence of the infection, the resistance of the individual attacked, and the pathway of the infection. In this regard the progress of the middle ear infection into the labyrinth is of considerable importance. Slow, erosive processes, destroying the labyrinthine capsule, are much more apt to arouse circumscribed intracranial complications than are labyrinthitides in which infection takes place suddenly through the windows. A notable example is the case reported by Thompson. In this instance cholesteatomata had eroded both labyrinths, and the latter were the seat of diffuse suppuration. Each lateral cerebellar lobe contained an abscess. The pathway of the intracranial infection was along the nerves in the internal auditory canal. That the pathway of the labyrinthine infection may be of importance in determining the character of the intracranial complication, even where the latter occurs with the acute otitides, the case reported by Uffenorde illustrates. Here the labyrinth became infected by an erosion through the promontory, and not by the windows.

Among unusual pathologic conditions associated with otitic cerebellar abscess, two cases in the series here reported are worthy of note. The one is an acoustic tumor reported by Dixon. The other is an abscess complicated by carcinomatosis of the temporal bone, reported by Hibler.

One of the most interesting problems connected with the pathology of brain abscess in general, but particularly with cerebellar abscess, is that concerning the process by which the brain substance itself becomes infected. All who have observed a number of cases of brain abscess have been impressed with the fact that in most instances the collection of pus is more or less remote from the cortex—i. e., that the majority of brain abscesses are situated in the white substance. To those who have performed postmortem examinations on a number of these cases, it has become evident that between the brain abscess and the source of the intracranial infection there is frequently a layer of brain substance which is macroscopically normal. In other words, there is a growing impression that the majority of brain abscesses do not occur through direct extension by continuity.

Even where there is a postpetrous epidural abscess, and where the brain surface is grown fast to the dura, there is often interposed between the epidural abscess and the brain abscess a layer of normal brain tissue. Thus, Isemer, in his case report states: "It is remarkable that, although the brain surface is grown fast to the dura in the region of the epidural abscess, yet between the abscess in the white substance and the area of dura there is a distinct, if thin, layer of macroscopically normal brain." From this one might conclude that the brain abscess was not the result of an extension by continuity from the diseased labyrinth, but that the infection extended by thrombosis of small cerebellar veins which empty into the pial veins, or through the lymph sheaths around these veins.

With regard to this question, Michaelsen (quoting Uffennorde) states: "The origin of the brain infection is always the meninges—i. e., the dura and the piaarachnoid. At first the pial veins become involved and thrombosed. This thrombosis which, in the beginning, is not always septic, follows the venous channels backwards, leaving the cortex intact, until it reaches the ultimate ramifications of the veins in the white substance, where the stasis produces red softening. The infection then follows and an abscess results. The cortex is not involved because of its separate vascular system."

Even though the "origin of the brain infection is always the meninges," yet the appearance of the dura and its behavior during the postoperative treatment, often give absolutely no indication as to the presence of a brain abscess. Collections of pus within the brain, with a macroscopically normal dura, are not so rare. In a case of right temporosphenoidal abscess recently observed by the writer, the dura was disclosed at operation covered with cholesteatoma and large pussy granulations. During the postoperative dressings the dura returned to normal—i. e., it became coated with small red healthy granulations, and healing was uneventful. Two months after the radical mastoid operation the brain abscess ruptured into the lateral ventricle, and death ensued from diffuse purulent meningitis.

The observations of Heilbronn, although too isolated to be conclusive in themselves, throw a flood of light upon this question. He states: "There are few observations of brain lesions which have occurred through venous channels (Rücklaüfigem Wege). Such affections we would expect especially in disease of the great sinus, the chief return current of the brain's venous blood. With the most frequent form of sinus disease—i. e., thrombosis and thrombophlebitis—there have been few observations of brain disease."

Oppenheim in his "Lehrbuch der Nervenkrankheiten" states that he believes there any many opportunities in cases of sinus thrombosis to observe focal brain symptoms. Hemorrhage and softening are often the direct result of sinus thrombosis, and, although such observations are rare, they probably occur far more frequently than they are observed.

Macewen reported convulsions in children which were confined to one side of the body, and which he attributed to thrombosis of the longitudinal sinus and the veins of the motor area.

Heilbronn reports several interesting cases with general and focal brain symptoms complicating otitic sinus thrombosis. In the first case, a patient with acute suppurative otitis media and mastoiditis, symptoms of meningitis followed six days after a simple mastoid operation had been performed. With a gradual rise of temperature the patient became delirious and finally comatose. Postmortem examination showed no meningitis. There was a thrombosis of the left sigmoid sinus. There were hemorrhages in both optic thalami, thrombosis of the venæ Galeni and of other basal veins. The symptoms were due to the disturbance in the intracerebral circulation. He draws the following conclusions: 1. In sinus thrombosis, by the plugging of the small veins, lesions of the brain in the form of hemorrhages and softening may occur. 2. These lesions, like many other brain lesions, may cause general brain symptoms without any definite focal symptoms.

The second case was that of a young man in whom a sinus thrombosis complicated a chronic suppurative otitis media. He suddenly became unconscious. This condition, however, lasted but a few hours, and was followed by an aphasia. Postmortem examination showed extensive sinus thrombosis. The large pial vein which surrounds the left temporosphenoidal lobe, and has its origin in this lobe, was thrombosed. The aphasia was due to the occlusion of this vein, and stasis of the blood with the consequent nutritional disturbance.

The third case, probably the most significant, was that of a young woman in whom a sinus thrombosis complicated a chronic suppurating ear. In this instance there were signs pointing to a bulbar lesion. Anatomically there was no gross obstruction of the veins leading from this region, but in the medulla there were changes which doubtless indicated a destruction of function of this part. There was destruction of fibers, a paucity of nuclei, while the remaining nuclei stained poorly and were deformed. The picture was one of anemic necrosis. The lesions were significantly distributed about the vessels. To the center of each focus ran a small vein.

These observations, while they are, as stated, too isolated to be conclusive, yet offer strong confirmation of Uffenorde's theory. Whether this theory, regarding the manner in which the brain tissue itself is infected, holds good only for those cerebellar abscesses which take their origin in a thrombosed sinus, or whether it can be applied to all cases, irrespective of their origin, remains to be proven. Only careful histologic research will clear this point.

In the eighty-six cases which we have collected the infecting agent is stated but eighteen times. The streptococcus was found eight times. The staphylococcus was found once. Among unusual bacteria may be mentioned the colon bacillus and Vincent's spirillum and bacillus. In their study of the bacterial agents in ten cases of intracranial complications of otitic disease, Turner and Reynolds found that streptococci were the virulent organisms in seven. In not a single instance of Michaelsen's series was the infecting agent in the cerebellar abscess found in pure culture. He found different bacteria at various times in the same brain abscess. Moreover, he found that similar changes occurred in the spinal fluid. Such results are unusual.

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V.

TUBERCULOSIS OF THE MIDDLE EAR.*

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During the last five years it has been my privilege to study nine cases of tuberculosis of the middle ear, which are given here with as little accessory history as is possible without vitiating their value.

Six were in children, three in adults. One of the children's cases, No. 6, is simply a clinical diagnosis with but little to confirm it, but represents a type which has been clearly defined; and as our diagnosis in all cases is seldom confirmed by every means possible, and in nearly every case some question may be raised, I believe I am justified in trusting my personal judgment of the clinical picture.

Two of the adult cases, Nos. 7 and 8, are diagnosed more by the course of the disease and the juxtaposition of pathologically tubercular tissues in otherwise healthy individuals: one (No. 8) showing a tubercular meningitis, the other (No. 7), a tubercular ulceration of the septum.

I believe that enough work has now been done on this subject to give to the profession a well defined clinical picture; so much so that the trained aurist should be able to venture a diagnosis of tuberculosis of the ear nearly as readily as the internist does that of tuberculosis of the lungs without a microscopic examination of the sputum.

The diagnoses are based upon:

1. Clinical histories and examinations.
2. Pathologic sections, which Brieger states is the most reliable and accessible.
3. Presence of tubercle bacilli in the secretion from the ear—a rare occurrence.
4. Animal inoculations (difficult on account of mixed infections).

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Case 1.—Family history unknown. Fed on a formula of cream and cow's milk since birth. Has always vomited food. Wassermann negative. Salvarsan.

At sixty-four days of age the cervical glands were enlarged, and pressure on these caused a discharge of pus from the external auditory canal. Temperature, 98°; hemoglobin, 64 per cent; polymorphonuclears, 62 per cent; lymphocytes, 31 per cent; large mononuclears, 6 per cent; transitionals, 1 per cent.

March 25th, at sixty-eight days of age, facial paralysis. Examination showed granulation tissue in left canal, much pus, enlarged cervical glands. Secretion from ear showed large number of tubercle bacilli. The child died March 30th, of an acute general tuberculosis.

This is one of the youngest cases on record. The lack of careful observation leaves much to be desired, but the inference is that the tubercular middle ear infection was of much earlier date than two months of age. The route of infection here is only problematic. It may have been through the blood stream from the lungs, as the child died of an acute general tuberculosis at the age of seventy-two days. On the other hand, the child was fed on a formula and probably had an infection of the bovine tubercle bacillus through the eustachian tube. This does not preclude the possibility of a lymphatic gland infection from the tonsils and then to the middle ear, but this is a rare route. The examinaiton of the tonsils in this particular case would not have thrown any light on the subject.

Case 2.—Two and one-half months old. Weight, six pounds fifteen ounces. Discharge from both ears. Cervical gland enlargement, fluctuating on left.

The radical operation on the right mastoid showed a granulating mass filling the mastoid and middle ear, with an involvement of the promontory and petrous portion of the temporal bone about the semicircular canals. The left mastoid involvement was not so extensive. The tissue section showed tuberculosis, and guinea pig inoculations gave a typical general tuberculosis.

The child died in the course of five months, the autopsy showing a primary tuberculosis of the bowel, miliary tuberculosis of the lungs and spleen, cervical and retropharyngeal lymph glands, and double tubercular mastoid.

In this case we have a good example of the fungoid form of tuberculosis, showing the eroding process on the promontory wall and about the semicircular canals.

Case 3.—Five months old. Breast fed for six weeks; cow's milk for two months, then Eagle brand. At two months child vomited persistently. At three months mother noticed an ear moisture, and child seemed in pain. This moisture and pain lasted for twenty-four hours, and since then there has been no discharge from the ear. The mother noticed an enlargement of the cervical glands at four months. On examination a smooth, pale tumor was seen in the ear canal. The glands of the neck were markedly enlarged on the diseased side.

Radical operation showed a caseous mass filling the middle ear and mastoid. This was removed and disclosed a promontory covered with granulations, part of which were removed. Filling the middle ear was a pear-shaped, smooth tumor which had eroded the floor of the middle ear and external canal, probably by pressure. On examination this proved to be a tuberculoma. Two years later the child was healthy, showing no signs of tuberculosis.

This is a remarkable example of the necrotic form proceeding to rapid destruction and caseation of the bone without a secretion from the canal.

Case 4.—Nine months old. Normal birth at full term. Weight, eight pounds. Breast fed for four months. At five months discharge from left ear. At seven months swelling about concha. At no time has the child seemed in pain. Temperature, 98.8°; pulse, 100; respirations, 20. White blood cells, 14,000; polymorphonuclears, 56 per cent; lymphocytes, 44 per cent.

A radical mastoid was done on January 5, 1914, after nothing definite had been made out in the lungs. The baby died on the 18th, of a general tuberculosis.

At operation a caseous mass was found involving the mastoid zygoma, middle ear, anterior wall of external canal. A sequestrum of the mastoid and posterior wall was removed. The dura was not exposed. Guinea pig inoculation of masses of granulation tissue removed at operation produced a marked general tuberculosis. Pus showed no bacteria, no growth on culture media. Sections of the tissue removed showed bone trabeculæ absorbed in many places, giving a spongy appearance. The marrow was largely replaced by endothelial leucocytes, and in areas granulation tissue. The endothelial leucocytes were massed in places and showed degeneration. In other places definite tubercles with giant cells were seen, and caseation was present. Diagnosis: Tuberculosis. Postmortem showed a marked bronchopneumonia, meninges slightly milky colored and edematous, but no pus. This case is the necrotic form.

Case 5.—One year of age. Breast fed for one month. At four months of age mother noticed that glands of neck were swollen. These were incised by a doctor, and afterward the ear began to run. Positive von Pirquet. Wassermann positive. On examination the ear was found filled with a polypoid mass and pus. This was removed together with pieces of bone. A radical mastoid was done, at which time it was found that the middle ear and mastoid region were filled with granulation tissue and sequestrated bone, involving the external auditory canal. A partial facial paralysis was observed after the operation, which cleared up within five days.

Microscopic sections showed newformed granulation tissue infiltrated with leucocytes. In some areas there was considerable collagen. Scattered through the specimen were well defined tubercles with giant cells. No caseation. Diagnosis: Tuberculosis. Child living and well.

This is a pitiful case of tuberculosis and syphilis combined, of the fungoid type, which is apparently well six months after a radical mastoid.

Case 6.—Aged seven years. Had had a slight discharge from the ear for two years, during which time there was no pain. The secretion was minimal. The drum was swollen, pale red, and did not change during the two years. Hearing poor. No nasopharyngeal trouble. The general symptoms were those of tuberculosis: loss of appetite, paleness of skin and mucous membranes, night sweats. Von Pirquet positive. For three years the ear has not troubled the child. Treatment was directed to the general health.

Case 7.—Twenty-eight years of age. Extremely good health; robust looking individual. Up to one year previous no ear trouble. At that time noticed an itching and stopped-up feeling in the left ear. No pain until two months previous, when severe over left side of head and in ear. Drum then broke and ear ran. Was in bed on account of "grippe." Was nauseated and vomited. Dizzy for a week, room going round severely for one week. No ringing in ear. Noticed she was deaf in that ear. One and a half weeks before my first examination there was pain in the right ear; three days later a discharge, continuing for one week. Hearing has been poor since then. There was no nose cold at that time.

Examination showed total deafness in the left ear and partial in the right; spoken voice, four yards; whisper, one yard. Weber to the right. Slight discharge in the left ear. Left drum shrunken. Perforation in the posterior superior quadrant. Right drum thick, pale yellow, bulging slightly, hammer lying in a furrow, arteries radiating over drum from center. No translucency. A paracentesis needle passed through an extremely thick membrane, as though there was no cavity behind it, and there was no discharge. Two days later a thick, yellow discharge, small in quantity; on third day, no opening. The nasal mucous membrane looked moist and normal. Posterior rhinoscopy showed a granulating ulceration on the posterior border of the septum in its whole extent. This, on being removed and sectioned, showed it was a tubercular tissue.

The right drum was watched for six months, during which time it did not change its appearance much; a couple of paracenteses were done, always with the same sensation of passing through a solid mass, and giving little or no secretion save on politzeration, when there was a thick mucopus noticeable.

The right ear in this case represents the type described by Jorgen Möller,[1] which he calls a myringitis tuberculosa diffusa. He excised portions of the drum and demonstrated tubercle giant cells.

In Cases 6 and 7 we have the picture of the infiltrating form: one occurring in a child and lasting two years, with other signs of tuberculosis in the body; the other a local affair in a superbly healthy and robust individual, showing a local tubercular lesion near the tubes. The rapidity with which the left labyrinth was destroyed by the process in the adult is uncommon, but the early labyrinthine degeneration in the right ear is frequent in this type of case and speaks strongly for its tubercular origin.

Case 8.—Aged thirty-five years. As a child had been considered scrofulous; in later life healthy. Lungs normal and no signs or symptoms of tuberculosis on careful search. Had had an ear discharge all her life. At no time had it been profuse; at times entirely dry for a month, then a slight moisture; never had it run out of the canal. The hearing had been slightly impaired in later life.

Examination showed a perforation in the posterior superior quadrant, extending well into the lateral attic wall and exposing the hammer neck. There was a crust over the perforated area and slight moisture on removal. On account of the history, a diagnosis of tuberculosis was made and an operation advised; this was delayed for seven months, when the patient fell into the hands of a general practitioner on account of a "general nervous breakdown." I saw the patient some two weeks after, when there was a marked meningitis, and operated the mastoid. I found a dark black, dry appearing bone, which blackness extended to the sinus. The cells were partly filled with a granulation tisue, partly with a dark, glistening mucous membrane. This pathologic bone was less marked at the apex than around the antrum and sinus. As I approached the sinus, a profuse hemorrhage occurred from the increase of small arteries in the chronically inflamed tissues about the sinus. This ceased when the sinus was well uncovered. The patient did not experience any benefit from the operation, and died two days later. An autopsy showed a marked tubercular meningitis, the tubercles being most pronounced in the sulci on the diseased side.

This is the only case I have seen of the lupoid form of the disease. The disease progressed slowly, in attacks, with little or no secretion or discomfort to the patient, to a tubercular meningitis by a path that was well marked at operation, and probably could have been checked by an early exenteration.

Case 9.—Aged forty-five years. This was a chronic case of tuberculosis of the lungs, which developed a double suppurative otitis media in the last three years of his life. There had been a deafness in the left ear for a year and a half. Dizziness a month before death, and deafness in the right ear, coming on suddenly. There had been a facial paralysis on the left side for some months. An autopsy showed chronic tuberculosis of lungs and ears. The left middle ear was entirely filled with a thick caseous material. The malleus and incus were destroyed, the stapes was still in position. The promontory was eroded but not perforated macroscopically. Smears from the caseous material and scrapings from the promontory showed numerous tubercle bacilli. Sections of the petrous portion of the bone showed the labyrinth to be completely filled with a caseous mass of granulation tissue showing an infiltration with polymorphonuclear leucocytes, tubercles, and giant cells of the Langerhans type. The spaces in the surrounding bone were filled with similar tuberculous granulation tissue. In the cochlea remnants of the organ of Corti might be made out.

This case is the necrotic form, showing the erosion of the promontory wall which is so familiar in tuberculosis. The process is more apt to extend to the labyrinth by a multiple perforation of the bony part than through the oval or round windows, as is the case in other suppurative processes. This explains the gradual labyrinthine degeneration and progress to deafness without the sudden labyrinthine symptoms in the old chronic cases.

Figures 1 and 2 are taken from this case. Figure 1 shows the infiltration of the petrous portion of the temporal bone, vestibule, semicircular canals and cochlea by the tubercular process. Figure 2 shows two tubercles and giant cells enlarged from Figure 1.

The incidence of tuberculosis in the ear varies so at different periods of life that no general statement is of any value whatever. Siebenmann, in a verbal statement to the author some years ago, claimed that the large majority of the ear suppurations in youth were of tubercular origin. I would modify that by making it in infancy. I cleaned out the adenoids and tonsils in all cases of ear suppuration in an orphan asylum, some twenty-five cases, ranging in age from five to fifteen years, and got an immediate cessation of the suppuration in all but two cases. These showed signs of general tuberculosis, and probably were of tubercular origin; the rest were undoubtedly a direct result of the presence of a pathologic condition about the eustachian tube. This does not occur in infants under one year of age; their ear suppurations, as a rule, persist after the adenoids are removed, leading us to the conclusion that an ear suppuration in these cases is due to some other cause than the adenoids.

Our autopsy statistics are worthless in estimating the frequency of the disease, as few autopsy surgeons ever even look into the external ear of their cadavers, let alone examine the middle ear or labyrinth. To illustrate: Wollstein and Bartlett have recently published the results of one thousand three hundred and twenty autopsies at the Baby Hospital of New York, covering a period of six years. Of these, one hundred and seventy-eight showed tuberculous lesions, ages from two and one-half months to five years; of these again, sixty-nine, or thirty-eight per cent, had a tubercular meningitis. As high as ten organs were involved in one case, but not once was the middle ear mentioned. In the light of my experience in a comparatively small clinic, I would say it was impossible to do autopsies on sixty-nine cases of tubercular meningitis in infants without having found a focus in the ear in at least two or three.

Until more careful observations are made at the autopsy table, we will have to depend upon our clinical and operative experience to draw any conclusion as to the frequency of this disease in either infants or adults. I believe its incidence in adults is much less than in infants, because of the high mortality in these latter cases; spontaneous healing appears to be a rarity, and operative interference not frequent.

It seems probable that in the ear, as in other parts of the body, the lesions are due to the actual presence of the tubercle bacillus, either bovine or human, and not to a chemical toxin generated by the bacillus in other portions of the body. Repeated attempts have been made to establish that the socalled scrofulous condition of the individual has been a factor in the production of ear discharges and of various nerve and labyrinthine lesions, but there has been no direct evidence, and most authorities look with suspicion on any such claims.

That the bovine bacillus is a frequent pronounced factor even in nursing infants is doubtful in my mind, because Ghon has proved through his large autopsy experience in Vienna, at the Kaiserin Elizabeth Kinderspital, that the disease in the vast majority of the cases is air borne to the lungs and is carried secondarily to other portions of the body. Tuberculosis occurring primarily in the digestive tract, as would be the case in infections with the bovine bacillus from the milk, are rare. This work has been confirmed by Wollstein and Bartlett in

New York, who, although not taking the extreme stand that Ghon does, approve of the general principle. In all of my cases where the possibility of a bovine infection is strong (Nos. 1 to 5), the children had received cow's milk for at least three weeks before any ear symptoms had been observed, but in those cases where autopsy was performed (Nos. 1, 2 and 4), in only No. 1 was there a predominance of intestinal lesions. For or against a path of infection through the tonsils, as might well occur in the milk borne infections, in these five cases I have no evidence.

The most reasonable path of infection for tuberculosis of the middle ear is through the eustachian tube, both for infants and adults. The constant presence of tubercle carrying phlegm, food and lesions about the mouth of the eustachian tube, together with the explosive coughs generally accompanying their presence, gives reason enough for the transmission to the middle ear without the intermediary of the blood or lymph stream. Blood borne infections do occur, and it has been well established that the tubercle bacillus circulates in the blood stream in lung infected patients; but were these infections the more common, there would certainly be more acute general or miliary tuberculosis among our patients with ear infections. Two of my infants are well after eradicating the focus in the ear, and one adult showed no signs of systemic tuberculosis.

The anatomic picture of tuberculosis of the middle ear varies widely and is evidently dependent upon the resistance of the patient and the length of exposure to the bacillus, as well as to the anatomic part involved. A patient that has been underfed, is congenitally weak, or lacks the resisting power for tuberculosis, will present a far different anatomic picture from a strong, well nourished individual, even though the lesion is limited to the drum membrane; so also will the early picture differ from a late one. In general, however, for classifying purposes, we can say that the picture varies according to the anatomic element: that is, there is a mucous membrane lesion, a drum lesion, or a bone lesion, giving three or more distinct pictures. Again, the process may be more or less general throughout the middle ear, and has been reclassified into lupoid, infiltrating, fungoid, necrotic, myringitis diffusa of Möller, and fibrinoid. Still again we have the acute and the chronic types. These classifications are more or less complicated, but are logical, and if once understood will be found not confusing in the least.

In Case 7 one finds in the left ear a comparatively acute middle ear infection leading to dull hearing for a number of months and an acute labyrinth destruction; the right ear became a chronic infiltrating type with a diffuse myringitis. Cases 8 and 9 were chronic, lasting in one case for thirty-five years.

The pictures become much more clear if we combine in our description the clinical with the pathologic. Those cases confined to the mucous membrane are, as a rule, infiltrating—that is, pathologically give a marked infiltration confined to the mucous lining of the middle ear—and clinically are subacute at times, leading on to an early perforation of the drum, but as a rule exhibiting deafness over a long period without secretion or perforation, but with a marked thickening of the drum. This thickening of the drum varies from week to week, and even varies in different parts of the drum at different times. The involvement of the hearing is often profound, and is not due alone to a thickening of the mucous membrane, but also to a labyrinthine degeneration, shown by functional tests and permanency of the deafness after the mucous membrane has returned to normal or has changed into the chronic suppurative type due to mixed infections.

The myringitis diffusa tuberculosa of Möller is an interesting form which he has observed as a pure type, but which is evidently a part of the infiltrating form.

Scheibe and others have described a fibrinoid form—which has not been found often, and of which but few studies have been made—in which there appeared a thick, dry, whitish yellow deposit on the promontory wall which was with difficulty removed to show a pale granulating surface on the bone. I saw one case which answers this description, but did not get a chance to study it. The fibrin that was removed did not show tubercle bacilli, and the case was lost track of without our getting a history. In this case the whitish deposit was leathery, and came away from the bone somewhat like a fungous growth from a tree.

The remaining types, lupoid, fungoid, and necrotic, involve all the structures, the lupoid being chronic, and the other two either acute or chronic.

The lupoid form, illustrated by Case 8, is one that appears in adults in attacks over a long period of time. The secretion in older cases is minimal when it does appear at all, and the destruction visible through the external canal is just as misleading. Brieger states that in the earlier cases, of which I have recognized none, the secretion may be pronounced during the attacks, the hearing becoming markedly involved only to return on a subsidence of the discharge. An ultimate labyrinthine destruction in these cases is frequent.

Of all the types, the fungoid and necrotic are the ones most frequently seen, mainly because they most often appear in children and infants; and again, they lead to marked suppuration and destructive processes, gland involvements, fevers and paralyses, which compel the attention of the parents or victims.

The fungoid cases present rapidly progressing granulation forming masses which appear in the middle ear and mastoid without producing much bone change, so that the skull is not entered, nor is there a great amount of pus. The drum often appears comparatively intact, and the small bones are removed at operation covered with masses of granulation tissue. The progress of the disease is rapid, the first symptoms often being a tenderness over the mastoid with a mastoid abscess. Often the middle ear is free, the process being confined largely to the mastoid alone. Cases 2 and 5 are of this type, and show the rapidly progressing character of the lesion in infants.

The necrotic form, illustrated by Cases 3 and 4, is more often present in adults, and progresses without the formation of the fungoid masses noted in the former form. In this class the pus formation may be extreme, and the antrum and mastoid filled with a caseous mass and sequestra floating in pus. The granulations lining the cavity are small and flat, and single polypoid masses, as were present in Case 3, are frequently encountered.

The fungoid and infiltrating forms may pass easily into the necrotic, but not vice versa. The latter remains pure throughout its course, and is much less likely to be checked by operative interference than is the fungoid. The infiltrating form does not lend itself easily to operative interference.

In the functional testing of cases of tuberculosis of the middle ear, one is nearly always struck with the disharmony between the remarkable deafness and the length of time and severity of the attack. The reduced perception for the voice and the low tones is of course the most marked factor, but the perception quantitatively for all the tuning forks is also markedly and rapidly reduced, so that one is reminded of the luetic cases he sees following salvarsan, suggesting that there may be some similar toxic effect in the labyrinth as well as a disturbance of conduction. In the later stages a complete deafness may occur without any vestibular symptoms having developed, and on sectioning a labyrinth we find a complete replacement of the labyrinth by a caseous or organized mass, as seen in Case 8.

The rapid erosion of the bone in the necrotic form and the infiltration of the tissues in the infiltrative form produce a facial paralysis much more often than do simple infections or infections of the lupoid or fungoid form. The paralysis in infants may be one of the earliest hints we have of any pathologic process going on in the ear. Alexander ascribes this to the frequent dehiscence of the facial canal, but others take issue with him and claim it is due to the peculiar pathology of these cases.

The diagnosis of these cases, outside of the history and physical findings, may be supported somewhat by the tuberculin tests and X-ray findings, but both of these are of minor importance. The tuberculin tests will be of more help in adults than in infants, where they are so often positive, regardless of an ear lesion.

Bernardini's work on the cytology of the secretions has become a classic, and his observation that the large mononuclear leucocytes are present in large numbers in tubercular middle ear secretions, lying in a clear, often fibrin holding serous fluid without pus corpuscles, is of immense value as an easy hint during our routine clinical work.

Of the complications of the disease, hemorrhages of the carotid and cavernous and sigmoid sinuses, sinus thrombosis and brain tubercle are rare, but labyrinthitis and meningitis are not infrequent. The labyrinthitis may be a tubercular process, as in Case 9; an acute suppurative process due to the mixed infection, as I suspect was the caes in the left ear of Case 7; or a plastic exudative process, in which case the deafness would be progressive over a long period.

The meningitis is, as a rule, a contact infection, as in Case 8. Briggs speaks of the possibility of an ear infection proceeding through the lymph channels from a brain tubercle, and it is interesting to note that Habermann[2] has since described such a case.

Concerning the prognosis in these cases, we would say that in our experience, in even the most hopeless looking cases, there is still a chance of an arrest of the local process; and in the lupoid cases in adults, an early surgical interference should be as successful as it is in ordinary suppurations of the ear. The necrotic forms in adults do not give as encouraging an outlook, as they appear in the advanced general tubercular cases, and any operative interference is poorly borne by such patients.

Since writing this article I have had the opportunity to study two cases of tuberculosis of the middle ear in which the lesions were principally apparent in the drums, and from which specimens were taken for pathologic examination.

Case 10.—Ed. C., patient in tuberculosis sanitarium, with a diagnosis of tuberculosis made microscopically from the sputum. One month previous he noticed a slight buzzing in the right ear. Since then a deafness has been noticed. Both deafness and buzzing have increased in intensity without any pain until one week ago, when he had a slight twinge of pain which lasted only a few hours.

On examination the right drum is found intact, beefy red, flaccid, bulging, hammer furrow noticeable, and arteries radiating over the surface. There is a slight moisture in the external canal. Weber to the right and watch heard at two inches.

One week later there was a perforation in the posterior inferior quadrant. One month later two small round perforations were present, the second one having appeared in the position of a former yellow spot on the drum. The secretion was minimal; patient did not know there was any; no pain. One month later the two perforations had coalesced and a part of the drum was removed, sections of which appear in Figures 3 and 4, showing typical fibrous tissue base infiltrated with lymphocytes and endothelial leucocytes. Definite tubercle with giant cells are seen scattered throughout the section: the whole shows evidence of degeneration.

Case 11.—W. B. Case in tuberculosis hospital with diagnosis of tuberculosis of the lungs. Came to clinic complaining of a ringing in the left ear; never any pain or discharge.

On examination the drum was found to be slightly yellowish red, not bulging, arteries markedly injected. The hammer was plainly visible; did not lie in a groove. One week later the drum was thickened, darker red and had two or three slightly yellow areas on the surface. A paracentesis gave no discharge, the needle passing through a thick surface into a cavity. The next day the opening had closed. One month later there was little or no change in the drum, and a piece was excised for examination. This relieved the ringing in the ear. The specimen showed the same fibrous struma as that of the former patient with lymphocytes and endothelial leucocytes, definite tubercle with giant cells and no areas of caseation. Figure 5 is taken from this specimen.

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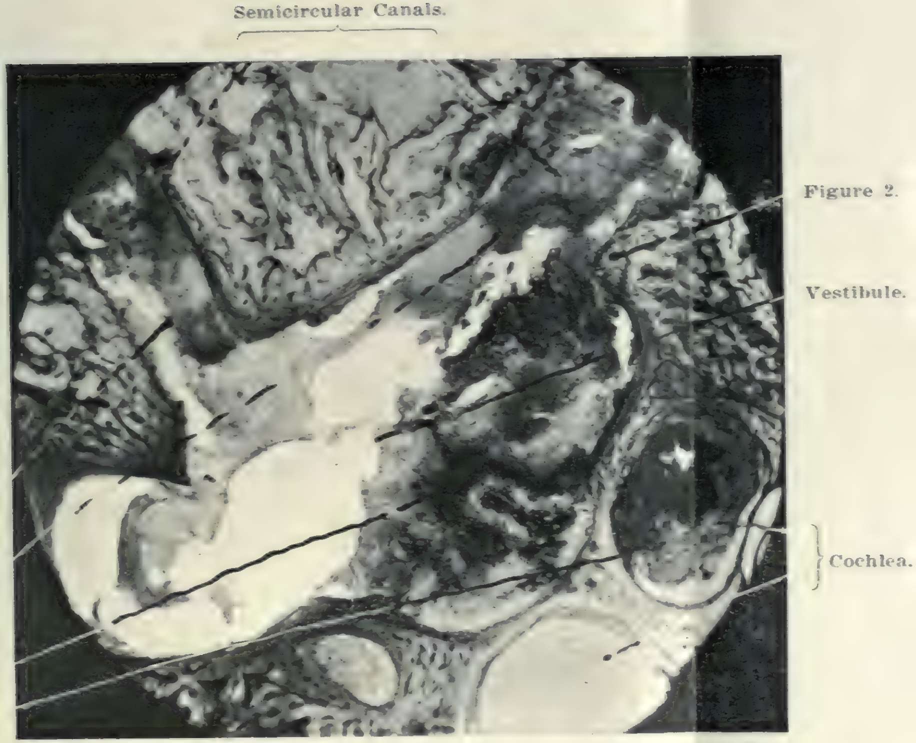


FIGURE 1.

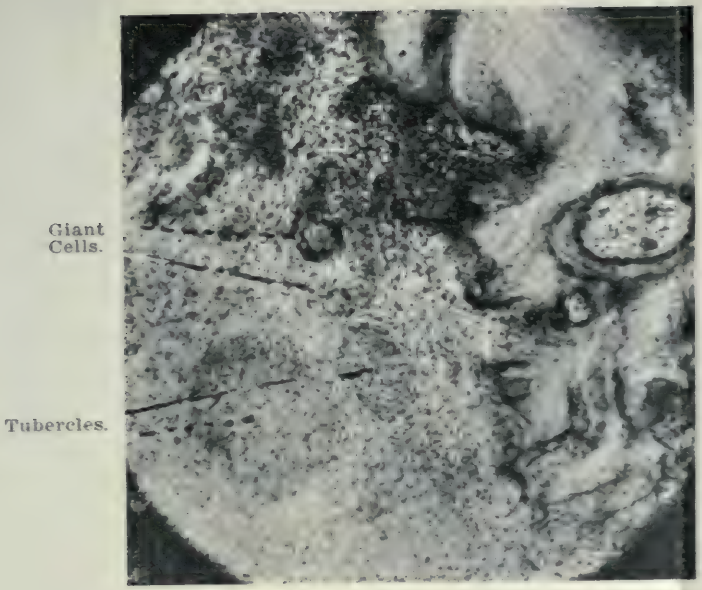


FIGURE 2.

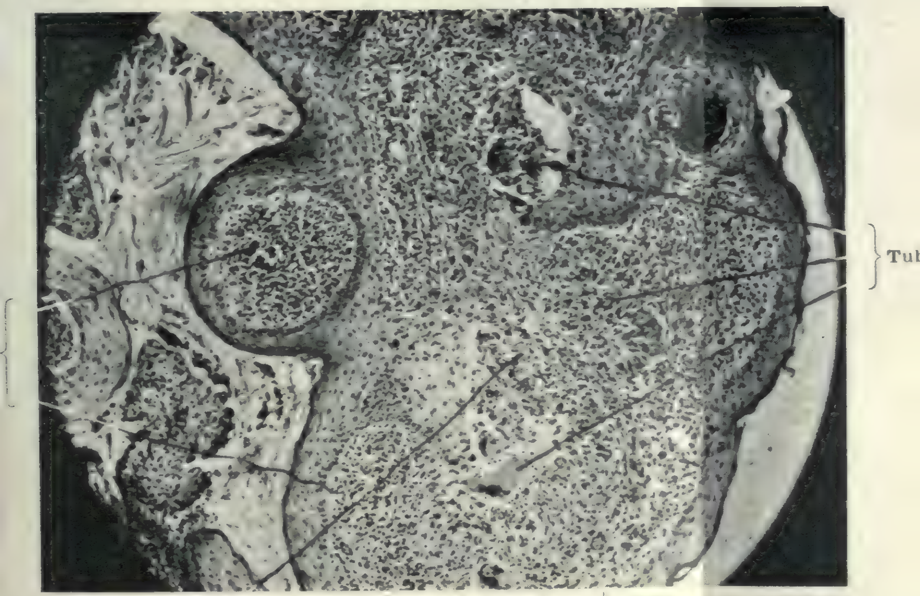


FIGURE 3.

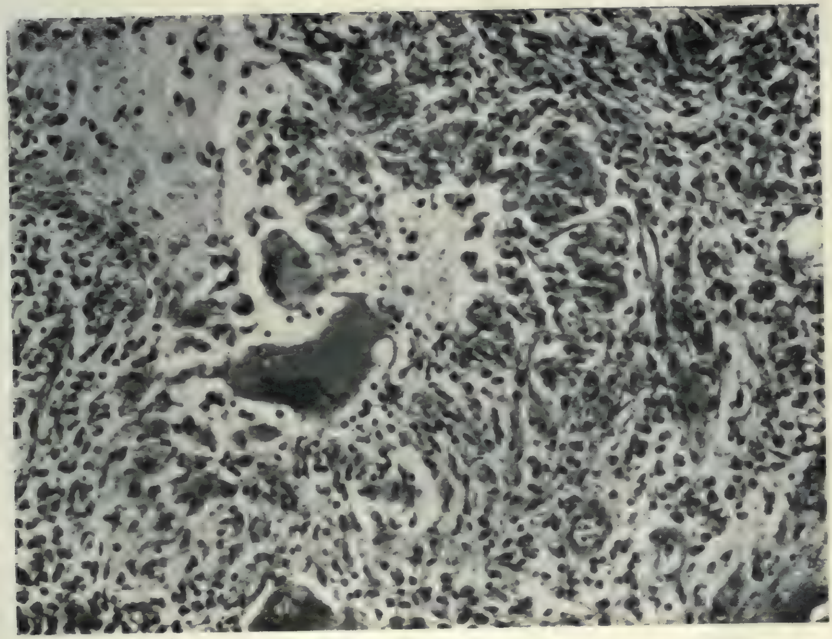
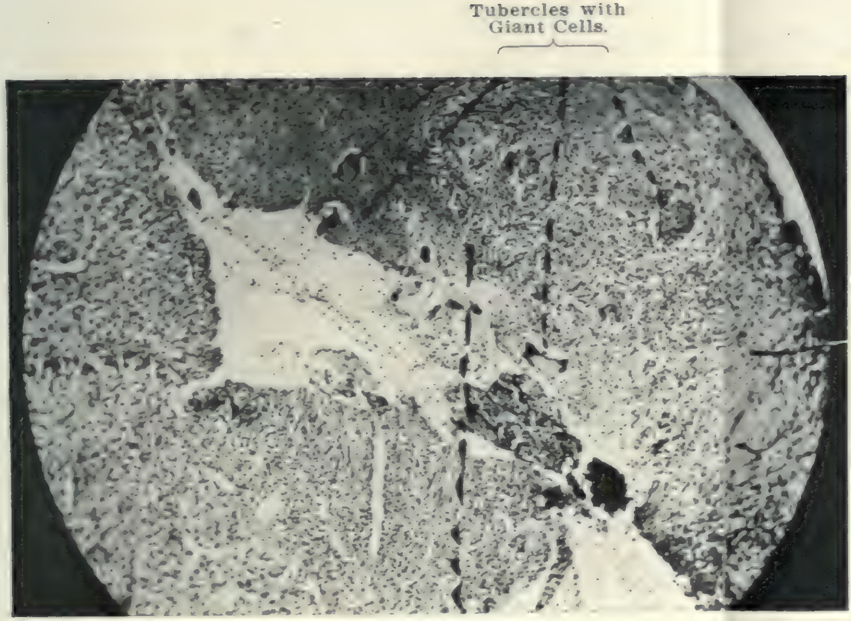


FIGURE 4.



Lymphocytes.

FIGURE 5.

IV.

BILATERAL PERIPHERAL PARALYSIS OF THE MUSCULI CRICOARYTENOIDEI POSTICI.*

By H. Edward Miller, M. D.,

St. Louis.

In this part of the world it seems to me that laryngeal muscle paralyses are less frequent than in the parts of Europe where I have been a student (Germany, Austria, London and Paris). But even where such lesions are more frequently seen, a peripheral paralysis of the cricoartenoideus posticus must still be a rare lesion, judging from experience and recorded instances. In addition, the case that I have had the opportunity of observing possesses some remarkable features, notably that three separate acute attacks have occurred in the course of simultaneous acute laryngitides. From the first there was complete recovery, from the second a trace of narrowing of the glottis remained, and from the third a distinct permanent narrowing followed.

N. H. F., a German, thirty years old, a practioner of medicine, whose history in every way is negative, consulted me March 25, 1906, for what seemed a moderate grade acute laryngitis of grip origin. Within twenty-four hours appeared unequivocally a bilateral posticus paralysis, beyond question of peripheral origin, a part of the acute process in the larynx. He became unable to open his glottis, and the dyspnea which followed was very great. His cords stood less than two millimeters apart posteriorly. Arrangements were made for an emergency tracheotomy, which was finally omitted because he improved. As the acute laryngitis subsided his cords began to open, and in three months his glottis appeared normal. He remained well for about a year, when he developed another acute laryngitis with paralysis of both cricoarytenoidei postici similar in every way to his first attack, except that he did not develop such high grade dyspnea, probably because he then understood better how to care for himself. The second attack was in every way identical with the first, except that a small degree of permanent narrowing of the glottis remained. A third attack, similar in every way to the first and second, developed in about a year and a half. Resultant from the last attack, remained a definite permanent narrowing of the glottis which measured about thirty per cent of its breadth posteriorly. Throughout all of these attacks there was only a slight huskiness of the voice, associated with the utterance of a few words, and rapidly followed by a noisy, labored and lengthened inspiration. Expiration was always quiet. Moving about or trying to sleep exaggerated the condition. Dr. Greenfield Sluder saw him in the third attack while I was absent from St. Louis.

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Since the last attack the patient has remained in good health. He has not manifested in any way signs of an organic central nerve lesion.

This case seems to me to merit whatever study may prove necessary for a satisfactory understanding of the various points of interest connected with it.

Anatomically the posterior cricoarytenoideus muscle is somewhat fan shaped, arising by a broad origin from the depression which marks the posterior surface of the cricoid cartilage on each of the crista. Its fibers converge in a direction upward and outward, to be inserted into the outer angle of the muscular process of the arytenoid cartilage. Its nerve supply is from the recurrent laryngeal, in common with the other intrinsic muscles of the larynx.

Functionally the cricoarytenoideus posticus seems to accomplish two results, one depending on its uncombined, simple action —i. e., acting alone—when it rotates the arytenoid cartilage chiefly around the axis of its saddle joint and at the same time slightly around its vertical axis. In this wise the processus muscularis is turned toward the median line and downward, and the processus vocalis outward and upward. During inspiration the cricoarytenoideus posticus widens the glottis, and performs a similar action when deep or soft tones are produced. In addition to its simple uncombined action, it may act in combination with its antagonist, the cricoarytenoideus lateralis. When both muscles act together the rotatory action of each is counterbalanced, and there results the downward and outward pull along the diagonal joint surface, producing a widening of the rima.

The symptoms of cricoarytenoideus posticus paralysis are quite characteristic. Since both vocal bands assume the median position, the glottis stenosis becomes severe, and in extreme cases the median border of the vocal bands approximate during inspiration. This narrowing of the glottis is explained in various ways. H. v. Ziemssen,[1] F. Riegel,[2] E. Burow,[3] and F. Semon[4] believe it a mechanical suction of the bands due to a negative pressure produced below the stenosis owing to the widening of the thorax during inspiration. O. Rosenbach[5] and H. Burger[6] assert that also in quiet respiration the adductors are innervated after the paralysis of the abductors, and that then the impulses are only in the direction of the normal group of muscles. K. Gerhardt[7] thinks both explanations plausible.

The inspiratory dyspnea is ofttimes associated with stridor and sonorous inspiratory noises. The voice is slightly husky, while the utterance of a few words is rapidly followed by a noisy, labored and lengthened inspiration. Moving about or trying to sleep exaggerates the condition. Expiration is easy and noiseless.

The diagnosis of cricoarytenoideus posticus paralysis is made by the omission of its function from the living larynx picture. It being the only muscle which opens the glottis, the cord cannot be moved outward when this muscle is paralyzed. A distinction must be drawn between the picture resultant from an arytenoid anchylosis from perichondritis with a cord fixed in the middle line, and a cord in this position from inability of the muscle to move it outward. In the former instance the line of muscle tension will be unchanged. In the latter there will be slight sagging outward, or maybe only flabbiness of the line produced by lack of tension; although this will be less than in the total cord paralysis of a recurrent trunk paralysis.

It seems to me that the diagnosis must always be made by the laryngeal mirror, although F. Riegel[8] thinks it almost possible without this aid. "However, often in tabetics the inspiratory dyspnea may be absent, and the mirror must then come into play," he adds. The cord stands in the phonation position. It might be pushed into this position by a growth from below the level of the cord, or an interarytenoid scar might pull the cords together; but such sources of error ought to be easily recognizable, as a rule.

In the consideration of such a case comes at once into mind the law announced simultaneously and independently by F. Semon[9] and O. Rosenbach (1880),[10] which has since become known as Semon's law—that the fibers supplying the abductors are more vulnerable than those supplying the adductors. So far as I can learn, however, this law is for trunk paralyses. In fact, I am unable to make out from the literature on this particular point that there is recorded a case of peripheral posticus paralysis. It seems to me that all the material discussed is of trunk origin. O. Chiari[11] emphasizes the fact that he had never seen a case in which he could diagnosticate posticus paralysis as being of peripheral origin. H. Tilley[12] records a case where a peripheral posticus paralysis may have been the lesion; but proof was not at his disposal.

The trunk of the recurrent laryngeal, with all its collateral bearings, I believe, is certainly to be excluded from this case because of the history of the three attacks and the behavior of the case for the three years which have elapsed since the last attack, the patient remaining in perfect health.

The socalled hysteric paralysis can also be excluded, it seems to me, from the course of the case over these six years' time. Also that none of the means usually successful with hysteric paralyses have had any influence upon it.

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VI.

THE CORRELATED ACTION OF THE PHARYNX AND SOFT PALATE, AND ITS EFFECTS UPON POSTNASAL DIAGNOSIS.*

By Greenfield Sluder, M. D.,

St. Louis.

By correlated action of the pharynx and soft palate must be understood the resultant action or effect of both the parts acting at once.

This district is complex anatomically, and serves in several functions—namely, swallowing, gagging, vomiting, sneezing, sucking, coughing, snoring, breathing, speaking, and singing—each of which is complex physiologically. My concern here is with the nasopharynx and oropharynx, and the functions of empty swallowing and gagging.

The simplest conception of the district, it seems to me, is that it resembles a soft walled tube, wanting in large part in front, where the soft palate, acting as a flat valve, divides it into a nasopharynx above and an oropharynx below. The upper outlet is fixed bone and divided in the choanæ by the septum nasi. The deficiency below is the posterior outlet of the mouth. The action of the tube in swallowing is a vermicular contraction from above downward, the anterior lower deficiency being closed by the tongue, which moves backward and upward, while the palate closes off the upper part. In ordinary swallowing a gentle degree of musclar contraction is used. In gagging the vermicular action is from below upward with the strongest contraction.

In or upon the posterior wall (pronounced in children), above the soft palate, is placed the pharyngeal tonsil, increasing in front to back measurement from below upward, its uppermost part a free surface, somewhat shelf-like, almost touching the basilar process of the occiput; or it is a little further removed from it, according to the degree of swelling or shrinking which has gone on in it. Laterally, below the soft palate, are placed the faucial tonsils, varying in size and degree of imbedding. They are nearly always limited below by their junction with the lymphoid tissue at the lower lateral aspect of the tongue, which is a rather constant point. Their limitations above are less constant.

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*Read before the American Laryngological Association, May 25th, 1915.

Two types of tonsils may be recognized—one placed free ("unattached" or "nonadherent") between the pillars of the fauces; the other placed deeply (imbedded) between them, little if any of its substance protruding "free" into the pharynx. According to the degree of enlargement of the organ, either of these types may rise into the mobile palate, even to meeting in the median line, compressing the uvula between them or crowding it forward and upward. When this is true, the upper lateral limit of the tonsil mass rises often to the junction of the upper and middle thirds of the palate; but this is less apt to be true for the free tonsil than for the imbedded one. The large "free" tonsil may lie under the palate arch in equal volume, but will push up the arch itself rather than ascend within it, which makes it (the arch) look higher, if not stand actually higher.

Just above the palate and slightly posterior to the middle line of the tonsil is found the cartilaginous mouth of the eustachian tube, which varies in size, prominence and position; being small and flat, or large and protruding one-half to one centimeter into the pharynx; or rising as high as the middle turbinate. Posterior to the mouth of the tube is the recessus pharyngeus (fossa of Rosenmüller), which may be scarcely at all marked or may be one and a half centimeters deep and one centimeter wide, extending outward and backward.

The free margin of the vomer begins below at the posterior limit of the hard palate and may rise straight up or pass upward and backward at an angle of forty-five degrees. The angle of the choanal plane is determined by this angle. Its vertical measurement varies from one and one-half centimeters to three and one-half centimeters approximately.

The plica septi varies in size and position, extending slightly beyond the free margin of the vomer; or is placed well forward of this, and sometimes higher or lower.

The soft palate is rather constant in length from its junction with the hard palate to its lower margin, but varies from two millimeters to one centimeter in thickness. The uvula and tongue vary greatly in size, and may play a part in correlated action.

The dimensions of the pharynx at the level of the palate may be five centimeters laterally, by two and one-half centimeters anteroposteriorly, which is the largest pharynx I have seen. Luschka[1] gives those measurements as normal. From my observations the average measurement in what seem normal adults in life is only about one-half of that size, and it seems to me that those measurements may be further reduced by one-fifth in what must often be admitted as normal. Such, however, must be considered a "small normal" pharynx. It may be narrowed to obliteration by enlargement of the structures found in it. The circular measurement of this plane may be greatly lessened, without the soft palate being shortened as it might be, were the simple requirements of the flap valve followed. It seems its length is determined by something else than its functional needs. Its elasticity may vary.

I have thought it of interest to know, for clinical use, what happens in the nasopharynx during the acts of empty swallowing and gagging, and to this end have conducted in a desultory fashion a number of experiments extending over thirteen years' time. No way was known to me by which these functions could be observed directly; and even now, with the help of Holmes' nasopharyngoscope and the X-ray, it still seems impossible. The X-ray of a bismuth bolus being swallowed shows its upward and backward, or its directly backward, course after passing the hard palate; but otherwise does not help. Einthoven[2] has given an extensive bibliography of writings on the physiology of the pharynx. But the particular type and purpose of the observations now to be described I fail to find recorded.

For my purpose I have tried many pigments to see if one could be found that would mark the surface wherever it touched, when placed on the upper surface of the soft palate in liquid form, one or two drops in volume. Two qualities were necessary in it—namely, that it should adhere readily to the membrane, and contrast sharply with it. These were found in a mixture of bismuth subcarbonat or hydroxid.

Nitrat of bismuth is dissolved in dilute nitric acid and mannite (about two per cent) added. To this is added a solution of carbonat or hydroxid of soda to neutralization. The precipitate is washed free of salts and allowed to settle, or centrifuged to the thickness of buttermilk. It is opaque white, contrasts sharply with the red of the membrane, and adherent to it under all conditions sufficiently to permit of any desired observation. Liquid India ink* may be used as a black pigment. A drop or two of the liquid is introduced through one nostril by means of a small caliber cannula and syringe, to arrive at the upper surface of the soft palate at a point in the center line of the choanal outlet. In a normad adult pharynx it is, upon swallowing, found spread over its posterior wall as high as the lower margin of the eustachian tube, usually equally on each side. The mouth of the pharyngeal bursa is below this level. In this way I have seen pus, secreted by the antrum of Highmore in small amount, come into the nasal fossa in some way that eluded detection, reach the soft palate from the floor of the nose, and be habitually absent from the nasal fossa, to appear spread over the mouth of the pharyngeal bursa simulating Thornwaldt's[3] disease. The soft palate acted as a trowel and spread the pus there as a mason spreads his mortar with his trowel. Inasmuch as no pus could be found in the nose, this deception was complete for some time. In this throat, gagging would have spread the pus in the same general way a little higher up. This effect, however, may be reversed. Instead of the pus being spread from the floor of the nose and soft palate, upward and backward, it may enter the nose from behind and above, and be wiped off the posterior pharyngeal wall to appear only upon the upper back part of the palate. Its origin then may be difficult to trace. In order that this may happen the throat must be narrow in anteroposterior measurement, regardless of what may be its lateral dimension.

Should the tonsils be small or absent, the loss of their pushing upward effect results in a lesser ascent of the palate. For the highest trowel effect in the nasopharynx the presence of the tonsils, to a greater or lesser extent, is necessary. The mouths of the tubes may or may not assist in this by the coming forward of the posterior wall of the pharynx to meet them, thereby obliterating the recessus pharyngeus. In a pharynx with small tonsils and small tube mouths, a drop or two of the bismuth mixture introduced to descend over the lateral aspect of the end of the lower turbinate will, in swallowing, be found spread on the lateral half of the posterior pharyngeal wall of that side at about the level of the lower part of the mouth of the tube.

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*Draughtsman's India ink is sold in the American markets in liquid form.

at about the level of the lower part of the mouth of the tube.

In the production of this picture the tube's mouth is set in the normal position—that is, there is approximately one-half centimeter free space between the tip of the turbinate and the cartilage of the tube; with like space between the floor of the nose and the cartilage, and like space in the recessus. The tonsils must be normal. Should they be large and rise high the pigment will be spread to a higher level, as they, also, are carried up in the rise of the palate and are assisted in this by the base of the tongue.

In gagging the contraction of the constrictors may be so strong that in a smaller throat the posterior wall of the pharynx is brought forward to the limit of the hard palate, and the lateral walls approximated. At the same time the soft palate is forcibly elevated to a considerable degree above the level of the hard palate, sometimes dimpling one centimeter above it. It should be recalled that the thickness of the soft palate is an average of about one-half centimeter, and frequently twice that; so this must be added in estimating, from below, the ascent of its upper surface. When the lateral walls are approximated and the posterior wall brought forward, and the palate is, as it were, stuffed upward into this narrowing gap, as happens in gagging, it is found that in the small normal throat the nasopharyngeal space is annihilated, as may be shown when a drop of bismuth mixture is placed near the center of the upper surface of the soft palate and gagging induced. The resultant picture shows the pigment to have been spread by a general approximation of the surfaces—that is, to be generally distributed—including an application to the free margin of the vomer. The angle of the choanal plane will play a part in this. This effect cannot happen in a large normal phraynx, or one of small normal caliber, but of unusual height, with a vertical choanal plane; for the tucking up of the palate is a necessary part, and it cannot be tucked up beyond its own length.

The parts of such pharynges cannot all be approximated, but with their diminution in size it becomes more and more easy, until it comes to pass in a small pharynx with normal swallowing. In this way I have seen seropus spread by the palate from the center of the floor of the choanal outlet, backward and upward, and lose all index to its origin. I have also in this way seen small amounts of seropus, entering the nasopharynx from above, wiped away and so spread out as to elude recognition. And I have often seen a sphenoidal discharge, profuse and purulent, wiped off the posterior wall, leaving no index to its origin, adhere to the palate and be raised over the tube into the middle meatus. At other times I have seen a small streak of thin pus, descending on the free margin of the vomer, wiped away by the palate and elude further recognition.

In considering diminution of size in the pharynx I feel that the means by which this may happen should be noted. A small pharynx may be part of a general diminutive state—e. g., childhood; or the older pharynx may be encroached upon in several ways. The pharyngeal tonsil may exist in greater or lesser bulk, narrowing its upper part from behind forward. The faucial tonsils may be enlarged, narrowing it laterally. I suggest scrutiny of the imbedded type, which may be large and not protrude beyond the surface. They are sunk into the walls and become evident only in gagging. I believe this type to be more potent in producing these effects than the prominent free one. It seems with the help of the tongue more efficacious in crowding the palate upward and backward. In gagging the tonsil masses are carried markedly upward in the strong upward pull of the palate, while the posterior wall comes forward.

The mouth of the tube—in usual position and environed by normal turbinates—permits pus, flowing backward out of the middle meatus, to descend over the lower turbinate between its tip and the mouth of the tube. The act of swallowing will then spread the pus or pigment on the same side of the posterior wall to about the level of the lower limit of the mouth of the tube or a little higher. In gagging, under these conditions, the pus or pigment may be transferred from the lowermost lateral point of the choana of one side to the uppermost lateral point of the opposite side—namely, the lower part of the sphenoethmoidal recess. This happens probably only when the contraction is strong in the obliteration of the last of the already narrowed space, after the palate has been forcibly elevated in a pharynx that is certainly not taller than normal (rather shorter).

The tube may be placed downward and forward of its usual position. Pus then proceeding backawrd from the middle meatus may pass over the tube backward and downward by way of the recessus pharyngeus. This is made easier by swelling of the posterior tip. In this instance the narrowing of the nasopharynx in swallowing, combined with ascent of the palate, may in propitious throats convert the recessus into a tube, which will have its vermicular movement from below upward. In this way pigment may be transferred from the floor or middle meatus of one side to the upper meatus of the same side, and this holds true for pus also. It may also happen that pigment or pus descending in the recessus from above is at times stuffed into the middle or lower meatus, provided it be caught in the right way to reverse this effect. Many times I have seen seropus, entering the recessus from above, by the act of swallowing, wiped away or so spread out as to elude recognition. I have sometimes seen, in gagging, pus from one olfactory fissure smeared over in such a way as to simulate suppuration of the opposite fissure also.

But in the production of these phenomena there seems to be little or no constancy. Sometimes the pigment will adhere more to the anterior parts, and at other times more posteriorly. This irregularity seems true oftener for pigment than pus. Possibly a difference in the moisture of the parts at different times determines this. Bismuth may be used to produce one picture, and then India ink, at another or the same point. In this way two experiments may be done in a throat at the same sitting.

Observation upon the suppurations leads me to believe that their pictures in the pharynx may vary—that is, be different one time from that which appears at another time under similar conditions in the same throat. And furthermore, that these pictures are not the result of a single act of swallowing. They are not so easily or readily produced as to result from a single act. It seems to me that pus is less easily transferred than bismuth, and that many of the pictures we find are the results of a small volume of secretion and repeated swallowing —that is, that a small amount of secretion is each time put into its unnatural position, and that the picture we see is the accumulated result of many acts of swallowing. Should the patient gag repeatedly in process of examination, these phenomena may be thereby likewise produced. Observation on the cadaver as well as on the living has shown me, moreover, that pus may enter the pharynx from different parts of the nose and not appear at the sinus outlet where it enters. Thin secretion may descend from the sphenoid sinus on the septum nasi anterior to the plica septi and not be recognizable by any of our present modes of investigation. It reaches the soft palate, below, and is then spread by its trowel action in such a thin film on the posterior wall that it is with difficulty or impossibly recognizable. The plica septi shuts it off from recognition by the postnasal mirror, and it cannot be recognized by the pharyngoscope. The anteroposterior distance between the osteum sphenoidale and the choanal plane varies much in different skulls. This explains how the last mentioned phenomenon may be a fact in some patients but not in all. The position of the osteum sphenoidale may sometimes be close to the median line, at other times placed far laterally.

A most elusive discharge is that which descends on the free margin of the vomer posterior to the plica septi, and is wiped off by the palate in swallowing.

The effects produced by a large tonsil on one side or a large posterior hypertrophy of one side may be calculated after one has done some of these experiments.

RÉSUMÉ.

To me it has been surprising to see the degree of narrowing (obliteration) that may take place in the nasopharynx in swallowing and gagging. This seems to have its limitations, however, in the soft parts. The elasticity of the tissues reaches a certain limit only. Therefore, when the distance between the posterior limit of the hard palate and the vertebral column is great—two and one-half centimeters, and the lateral measurement five centimeters—the combined narrowing effects are insufficient to obliterate such a large cavity in even the strongest gagging. The soft palate is not long enough to rise beyond the level of the tubes, and the loose tissue beneath the constrictors is insufficient to permit enough movement toward the center to result in approximation (obliteration).

But the degree of elasticity in the soft parts is practically constant. Therefore, when the nasopharynx is small either because of scant space in the bony skeleton or because of the size of the structures developed upon or within its walls, approximation of its surfaces (obliteration) is possible by the easy play of the muscles in swallowing or gagging. Naturally the smaller the pharynx the easier may it happen. In a cavity a little smaller than the normal, this would happen only upon strong gagging, and the pigment or pus would have to be in fair volume in order to be spread out; whereas in a small cavity a small amount of pigment or pus is spread out far, because the closing pressure is enough to spread a small amount over a wide area. This becomes easier until, in the small pharynx which one often sees in adults, not to speak of children, it may happen in tranquil, empty swallowing. The quality of the pus also makes a difference. Thick mucopus holds together and does not transfer readily, whereas seropus spreads easier.

Clinically, the understanding of these phenomena is of value —I believe especially so—when some other facts are borne in mind. In considering the transferrence of secretion from one place to another, thereby giving rise to clinical deceptions, it should be recalled that it requires a fluid secretion to permit of this, and that then it is easily done, and that the drier the district and the thicker and more tenacious the secretion the less likely it is to happen. Under the last conditions I have repeatedly tried, in vain, to bring it about. But of greater value, it seems to me, is to know the fact that by virtue of the power of the small pharynx to approximate its walls it possesses the power to wipe away small amounts of thin secretion from any point of the choanal plane, and even points forward of this (as may be shown by the bismuth being often put well into the meatuses). It is then so spread out over the general surface that it is lost from detection, and this may happen in the small pharynx in simple swallowing.

PRACTICAL APPLICATION OF THESE OBSERVATIONS.

Every rhinologist is familiar with the difficulties that attend the examination of some patients. Despite careful approach, every attempt to see the nasopharynx is followed by gagging, and even a palate hook in a small pharynx does not prevent obliteration of the fault, the posterior wall coming forward. When it is recalled that this simple act may create deceptions in any one of the above enumerated ways, their practical value is self-evident, and when one remembers that in small throats these effects can be produced not only by gagging but by common swallowing, it becomes at once evident that there may be certain nasal disorders requiring diagnosis from the pharynx that may elude recognition indefinitely—i. e., sphenoidal (and postethmoidal) discharge of this consistency descending on the posterior margin of the vomer, or anterior to the plica septi, or in the recessus or near the median line. Such discharge often cannot be seen from in front, including use of Holmes' nasopharyngoscope; so that when it eludes the surgeon from the pharynx, the deception is complete, and I believe may elude him indefinitely. This secretion from the sphenoid looks like thin cow's milk. For experimental reproduction of sphenoidal suppuration of thin consistency I have used thin milk or thin bismuth. It is very easily visible at the time of its instillation, but the last trace rapidly disappears from sight in the nose, and from a pharynx that wipes itself in swallowing. I have seen it lost in three minutes and leave no evidence to the patient's sensation—that is, not be recognized as having flown into the pharynx.

In a recent paper dealing with obscure headaches, I emphasized the fact that sphenoidal inflammation could lose the classical picture—namely, the easy showing of pus at the proper point—and continue indefinitely, making headache, but eluding recognition locally.[4] This happens because the secretion gets thinner. The above observations bear directly on such cases. Shrewd observers have emphasized the difficulties besetting sphenoidal diagnosis—conspicuous and familiar to all of us is Dr. Casselberry's paper before this association in 1911.

When one bears the possibilities in mind—or, better still, will try the various nasopharynges experimentally—he soon learns to estimate—and I believe with reasonable accuracy—the possibilities or probabilities of the individual one at the time of examination, and thereby may avoid some deceptions in the diagnosis.

In concluding, I desire to reiterate and emphasize that these effects can only very rarely be brought about in what I have defined as the normal adult pharynx with the exception of a simulation of Thornwaldt's disease, where the trowel action of the soft palate can do this. Very rarely the soft parts may possess unusual elasticity or freedom of motion. Such conditions may make it possible for the normal adult pharynx to approximate its walls not only in gagging but even in swallowing. Alterations in the form and size of the pharynx make the various deceptions possible, as herein enumerated. The pictures are produced with surprising clearness by bismuth or India ink, and much less so by pus. It is, of course, self-evident that pus at the classical sites, well forward of the choanal plane, with or without a clean pharynx, is of conclusive diagnostic value, and is uninfluenced by any of the above enumerated possibilities; but he who must needs have these classical pictures clear, before he can make the diagnosis, will have been eluded by other positive cases.

I believe, in a case where the history of nose disease is reasonably clear and the diagnosis very difficult, that it is at times distinctly helpful to know to what extent the nasopharynx is obliterated in swallowing and gagging. This would seem evident from the above context, and may be learned readily by the bismuth experiment.

The deceptions besetting postnasal diagnosis of the suppurations in children are largely, if not wholly, explained by these observations.

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VII.

TREATMENT OF HEMORRHAGE WITH NORMAL BLOOD SERUM.*

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The arrest of .nonoperative hemorrhage, while not often coming under care of the otorhinolaryngologist, becomes a serious problem in the cases where total exsanguination is threatened as the result of incontrollable oozing from the mucous surfaces of the upper respiratory tract. A case of purpura hemorrhagica which came under the author's observation through the courtesy of Dr. ———, in whose service at the New York Postgraduate Hospital the author is working, together with two cases seen by the author and which were controlled by means of human blood serum, caused him to review the serotherapy of hemorrhage with special reference to its application in otorhinolaryngologic practice. As pointed out by Matas, this mode of treatment, while not specific in character, is based on a rational principle, and has yielded sufficiently encouraging results since its introduction to deserve further trial, not only in hemophilia, but in all the allied hemorrhagic states.

Serotherapy is based upon a physiologic observation that blood, having been rendered noncoagulable for experimental purposes, frequently recovered its coagulability when fresh normal serum from another animal was added to the contents in the test tube. Injections of fresh serum have since been found to possess a curative as well as a preventive value in the treatment of hemophilic bleeding, and it seems rational to resort to them in hemorrhage of any type. The organism in these cases endeavors of its own accord to increase the coagulability of the blood as a defensive act. The deficiency in the

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production of fibrin ferment in these patients is supplemented by introducing into the blood stream a substance in the form of a normal blood serum, which assists the organism to fabricate a new supply of fibrin ferment. The effect of the antitoxic serum is explained by the fact that fresh diphtheritic serum also contains the fibrin ferment lacking in blood with abnormal coagulation.

This method dates back some twenty years, when serotherapeutic measures were employed, although in a somewhat empirical and rather desultory fashion. The most brilliant modern results are probably those reported by Welch, who states that the subcutaneous injection of normal human blood serum will control the hemorrhagic diseases of the newborn in practically all the cases.

A brief review shows that in 1897 Bienwald, in Germany, succeeded in arresting by means of fresh serum a traumatic hemorrhage in a three-year-old hemophilic child. About the same time a case of purpura cured by fresh serum was reported by Felz and Pigot, in France; and in 1898 Fry, in this country, reported three cases of hemophilia in three brothers who were successfully treated with injections of horse serum. In 1907 Weil, in Paris, published his studies of hemophilic blood and the effects of serum treatment. He originally used fresh beef serum (now abandoned), fifteen cubic centimeters of which he injected into a woman suffering from purpura, subconjunctival hemorrhage and hematuria. The patient left the hospital well, but suffered a severe reaction after the injection, with chills, fever and vomiting. In the case of an alcoholic having chronic purpura with almost continuous hematuria, three intravenous injection of fifteen cubic centimeters beef serum controlled the purpura eruption and hematuria. Another case, a boy of nineteen years of age, with profuse epistaxis and hematuria, was given an injection of ten cubic centimeters fresh rabbit serum, which promptly checked epistaxis and diminished blood in urine. These observations were followed by many others.

To Weil is due credit for pointing out the prophylactic value of serotherapy. This preventive action is obtained from twenty-four to forty-eight hours after the injection, and persists for at least three weeks, often lasting two or three months, when the blood will be found to have again the former anomalies of coagulation. This point is of special importance in operative work on patients having hemophilia or purpura (manifest or latent).

Fresh normal sterile serum contains all the desirable fibrin ferments for the arrest of dyscrasic hemorrhage, and nothing else. For this reason it would seem preferable to the antitoxic serum. The human serum used in cases reported by the author was obtained fresh from the laboratory of the New York Lying-In Hospital, and used within forty-eight hours. The method of obtaining serum is the same as that in use for obtaining blood for the Wassermann test. Apparently healthy mothers with history of previous normal pregnancies and children are selected. The serum is obtained by allowing blood to clot in test tube and decanting the serum. An observation by Dr. A. B. Davis of the staff was to the effect that the serum obtained by centrifuging blood was not as active as when allowed to clot in the test tube.

However, the use of the antitoxic serum in cases reported by Weil and others was followed by beneficial results and no sequelæ of any kind, either immediate or remote. Human, rabbit, horse and beef serum have all been shown to be efficient in test tube experiments, and yet some consider the superiority of human serum for the correction of blood anomalies as quite marked. According to Clowes and Busch (1913), human serum is in no way superior to that of a variety of animals, notably the horse. The health of the giver in use of human serum must be investigated, and some prefer a near relative of the bleeder. In an illustrative case reported by Traver, a child of five years, of hemophilic ancestry and himself a bleeder, bled profusely from a small wound of the tongue; horse serum injection negative. The hemorrhage was controlled at once by injection subcutaneously of twenty cubic centimeters of serum from the father's blood. Human serum has the great advantage of protecting against anaphylaxis, no matter in what dosage it is employed.

Normal serum possesses a natural stimulating action upon the various cellular elements, such as the leucocytes and red corpuscles of the blood. Animals receiving serum injections are livelier and more active sexually, and there is improvement of the muscular power. Welch emphasizes the nutritive value of homologous serum, and refers the favorable results to the nutritive influence of the serum upon the endothelium of the blood vessels. The coagulation of the blood in hemophilic patients is not regularly accelerated through serotherapy, although the hemorrhages may be diminished, suggesting, perhaps, that the endothelial lining of the intima also plays a part in hemophilia. Serum, more particularly antitoxic serum, has been administered internally in milk. When used in this way, anaphylactic symptoms are less likely to result, and the introduction of colloid factors through the skin is avoided.

The possibility of transmitting syphilis must always be kept in mind, and this must be considered a disadvantage, and the only one. Rabbit serum is very slightly toxic for man, and can be easily obtained from the carotid artery under antiseptic precautions. The quantity obtained is only sufficient for one injection. Horse serum is obtained in large amounts, and seems to yield more uniformly satisfactory results than any other animal serum. Beef serum has a tendency to induce anaphylactic symptoms in the form of chills and fever, cyanosis, vomiting, headache.

For the checking of dyscrasic hemorrhages in an adult the intravenous injection of ten to fifteen cubic centimeters of blood serum has been used. In cases reported the subcutaneous use of twenty and forty cubic centimeters was employed, repeated in eight, twelve or twenty-four hours, depending on condition and results. Subsequent injections may be given over a period of several months. In children, half the dosage may be given and repeated as suggested. To obtain full therapeutic action serum older than two days should not be used. Serum should be kept in ice box and sterile.

Precipitated blood sera, in the form of serum powder, have yielded good results in hemorrhages after tonsillectomies and turbinectomies. According to Clowes and Busch, the serum powder has been applied as a local styptic to bleeding surfaces, and has also been used with apparent success as a prophylactic before operations upon the nose and throat. This is of special interest to the otorhinolaryngologist. Relatively large doses are given the day preceding and an hour before operation. Coagulose, now offered to the profession by Parke-Davis Company, and originated by Clowes and Busch, consists of precipitated normal blood serum, and is claimed to retain its active properties unimpaired for long periods of time. It comes in the form of sterile soluble powder in a sealed tube. Properly prepared, it can be used subcutaneously as well as locally in its powdered form.

Different opinions prevail as to the efficiency of coagulin Kocher-Fonio, which has been employed with satisfactory results, according to Kausch, in Berne. The powder is obtained from mammalian blood. Coagulin acts through accelerating the normal process of clot formation, and was usually employed externally on cotton swabs soaked in a solution. It has also been used intravenously. No harmful results have been thus far observed. Albrecht recently reported its use in gynecologic practice. In order to guard against ever present danger of secondary hemorrhage after endonasal operations, Barth, in a recent contribution to nasal surgery, recommends the use of coagulin Kocher-Fonio, according to the following method: Immediately before use, ten per cent watery solution is prepared and sterilized. After operation the wound surfaces are moistened with solution. Hemorrhage is controlled in nasal, adenoid and tonsil cases. Can be used as spray. There are no after-effects, and the repair process, if anything, is hastened. Coagulin was also employed for hemostatic purposes by Dr. von Lang, Budapest. He used a twenty to thirty per cent solution, applied on a strip of gauze, for two or three minutes. There was no secondary hemorrhage noted, and it was especially useful in operations of septum, ethmoid cells and antrum.

Serotherapy has been used by Wirth as a local application. Horse serum was used to control hemorrhage from the nose and mouth of a newborn infant. The bleeding spots in the mouth were painted with swabs soaked in the serum, and two wet gauze strips were inserted very loosely into the nostrils, with the result that the hemorrhage stopped.

The immediate availability and the advantage of permanence of action must prove the value of dried serum. It is well to note, however, the occasional failure of both fresh and dried sera. Claybrook reports a case of a child, fifteen months old, dying from hemorrhage of the tongue following an accidental cut by the teeth. Normal serum was injected subcutaneously, and calcium chlorid was given by mouth.

The subject cannot be dismissed without reference to use of whole blood. The transfusion of whole blood has been used both in this country and abroad. A physician in this state saved his daughter from bleeding to death in a post-operative hemorrhage following tonsil removal. All treatment failing, direct transfusion of whole blood from father to daughter was done, with resulting control of hemorrhage. Recent reports show the work being done to save life in cases of severe hemorrhage by Dr. Alexis Carrel, so adept in vascular surgery, through method of direct transfusion. The objections to direct transfusion given by Welch are, in addition to the technic difficulties of the procedure, the danger of hemolysis, thrombosis and embolism; all of these, save hemolysis, have been overcome through the simplified technic of Dr. Lindeman of New York. Before performing direct transfusion, in addition to negative specific findings, it is desirable to ascertain that the serum of the giver does not agglutinate the red corpuscles of the recipient, or vice versa. A serviceable method is recommended by Fischbein. The good result of direct transfusion in cases of hemorrhagic diathesis is explained by Duke, who found reduced number of platelets, the introduction of new platelets presumably supplying the necessary element. Cases reported by him showed relief from tendency to bleed in two cases of purpura hemorrhagica. Flörcken reports a case of dangerous hemorrhage from the nose, gums and gastrointestinal tract, in a woman, twenty-four years of age, without a hemophilic family history, in which, after all other measures had failed, including gelatin and diphtheritic serum, direct transfusion was employed with immediate and favorable effect. The donor was an older sister. In pernicious anemia, Flörcken found only transient benefit. In a case of melena neonatorum reported by Misenthal, direct transfusion arrested not only the hemorrhage, but created normal condition of the blood. It may be of interest to remember that according to Lindeman, direct blood transfusion is of benefit in septic patients, thus replacing elements that inhibit bacterial growth. Whole blood has been used subcutaneously.

Recently V. Horsley has suggested and used the application of living tissue to bleeding surfaces. This does not seem practical or necessary.

In addition to my review of serotherapy and direct transfusion, it has seemed advisable to add a few other recent cases illustrative of this line of treatment to those personally presented by me.

Case 1. Purpura Hemorrhagica.—Treatment, human serum injection. Male, aged forty-seven years. Admitted to service of ————, April 2, 1914. Severe bleeding from nose, ears and gums.

Family history: Negative.

Personal history: Inflammatory rheumatism, ten years ago. No other severe illness. Habits good. Lately worried and not sleeping well. Ulcer on right septum of unknown origin for several years. Nose bleed began two years ago; three attacks, severe, all from right side. November 12, 1913, eleven attacks in one week, controlled by (Dr. Spann) using adrenalin. March 13, 1914, slight attack. March 30, 1914, bleeding slow but persistent, from nose and throat and ears and gums. Spots appeared on body; later found to be subcutaneous hemorrhage. Hearing right ear affected.

On admission: Temperature, 104°; pulse, 104; respirations, 24. Bleeding from nose, throat, gums and ears. Purpuric areas on body. Throat culture negative to bacillus diphtheriæ; streptococci present.

Blood: Erythrocytes, 3,640,000; leucocytes, 11,400; hemoglobin, 68 per cent; polymorphonuclears, 81 per cent; lymphocytes, 18 per cent; transitionals, 1 per cent. Given forty cubic centimeters fresh blood serum in deep muscles of back; calcium lactat, grains five, every four hours.

Urine: Phenosulph. test: Amount secreted first hour, negligible; amount secreted second hour, 25 per cent. Total in two hours, 25 per cent.

Fragility of red corpuscles: Degree of hemolysis of corpuscles mixed with salt solution:

Percentage of salt: 0.3—0.35—0.40—0.45—0.5—0.6—0.7.

Degree of hemolysis: Complete—Complete—Moderate—Slight—None—None—None.

Fragility of cells not increased, but within normal limits.

April 3rd. Temperature, 103.4°; pulse, 92; respirations, 18. Bleeding intermittent, but severe all night. Severe hemorrhage early in the morning from mouth, nose and ears. Clot and bright red blood. Nine o'clock in the morning, forty cubic centimeters serum; still severe hemorrhage. Two-thirty o'clock in the afternoon, forty cubic centimeters serum, adrenalin packing of nose removed and replaced. Seven-thirty o'clock in the evening, still some oozing, but not as severe. Given fifty cubic centimeters serum; sulp., grain one-fourth. Temperature, 104°.

April 4th. Oozing stopped; fairly comfortable night. Temperature, 103.8°. Two o'clock in the afternoon, twenty cubic centimeters serum. Urine negative.

Blood: Erythrocytes, 2,510,000; leucocytes, 14,600; hemoglobin, 42 per cent; polymorphonuclears, 89 per cent; lymphocytes, 11 per cent. Wassermann negative; blood culture negative; coagulation time, ten minutes.

April 5th. Temperature still high; some oozing; eye and face puffed; finger oozing; coagulation time, three and one-half minutes. Culture of throat showed bacillus diphtheriæ, and patient was given 10,000 units. No further growth found.

April 7th. Improving. Blood shows several myeloblasts.

April 16th. Continued improvement. Blood: Erythrocytes, 2,450,000; leucocytes, 8,400; hemoglobin, 43 per cent; polymorphonuclears, 80 per cent; lymphocytes, 18 per cent; transitionals, 1 per cent; mast cells, 1 per cent.

April 23rd: Discharged, cured.

Case 2. Control of Nasal Hemorrhage by Human Blood Serum.—Male, aged twenty-four years. Referred to author by Dr. ———— by telephone, stating case as desperate from four days' bleeding from nose. Admitted to hospital November 5th. Clots in nose and nasopharynx, ulcer right side septum. Given forty cubic centimeters fresh serum subcutaneously. Serum obtained from laboratory of the New York Lying-In Hospital.

November 6th. Apparently no fresh hemorrhage. Given forty cubic centimeters serum and repeated next day. The patient had an active tubercular process in lung.

November 25th. Dr. Murray reports no further hemorrhage, but notes immediate beginning improvement in patient's general condition following use of the serum.

Blood Report in This Case.—November 5th, coagulation time, ten minutes. Erythrocytes, 4,204,000; leucocytes, 9,800; hemoglobin, 48 per cent. Differential—Polymorphonuclears, 78 per cent; neutrophiles, 75 per cent, and transitionals, 3 per cent; lymphocytes, 22 per cent; large, 10 per cent, and small, 12 per cent.

Case 3. Alcoholic Cirrhosis Liver, Purpura, Severe Hemorrhage.—Male, aged fifty years. Was seen by author with Dr.

————————. On November 15, 1914, patient was seized with attacks of hemorrhage from nose following alcoholic excess. Bleeding continued for two days and was followed by hematemesis on November 18th and purpuric areas. On the 19th hemorrhage became alarming. Fresh serum was obtained from laboratory and forty cubic centimeters given at eight-thirty that evening. This was repeated on the two following days. There was no hemorrhage after the first injection. Lindeman speaks of the use of blood transfusion in cases of alcoholic cirrhosis of the liver with severe secondary anemia.

ADDITIONAL CASES.

Case of purpura hemorrhagica, treated by horse serum. Dr. Bodenheimer. Given ten cubic centimeetrs of horse serum and repeated. No return of symptoms at the end of three months.

Case of purpura hemorrhagica, with spontaneous bleeding from nose and gums, controlled by antitoxin serum. Dr. Goldstein.

Case of nasal hemorrhage following turbinectomy in a hemophiliac treated by injection of human blood serum. Reported by Dr. Linn Emerson. Man, aged thirty-one years. Use of antitoxin serum at first, but not controlled. Serum obtained from patient's brother and friend. Large amount used and extended period of time must be noted.

Morestin reports a severe and apparently hopeless case of purpura, in which the patient was actually brought back to life by two injections of twenty cubic centimeters of normal horse serum, according to Weil's method. Wilson reports a case of Henoch's purpura successfully treated by injection of human blood serum obtained from the father's blood. Obstinate epistaxis in a hemophilic boy, arrested by twenty cubic centimeters serum is reported by Lommel. Weil distinguishes two forms of hemophilia—the accidental or transitory, and the congenital. The anomalies of the blood in purpura seem analogous, although not identical, with those of hemophilia. The use of serum is followed by a diminution, but not a disappearance of the blood anomalies. The toxic factors continue to impair the blood in the progressive cases, and here the results of the use of serum are naturally only transitory. Welch claims one hundred per cent cures in cases of hemophilia neonatorum at the New York Lying-In Hospital. Treatment should be begun at the slightest suggestion of hemorrhage. Epistaxis due to arteriosclerosis was favorably influenced by serum injections in the experience of Wirth, who reports a case of a male, aged fifty-seven years, in which serum injection and serum tampons to the nose were used. Ramond recommends serum injection in grave cases of hemoptysis.

Injection of blood serum as a prophylactic measure is taken up by Goldstein, who advocates the method as a most potent and valuable ally in the control of hemorrhage, and emphasizes its advantages. The coagulation time is systematically tested in each case of tonsil enucleation, and whenever it exceeds seven minutes an injection of serum is administered. Fleischer in 1911 spoke of the use of serum at the first Northern Oto-laryngological Congress, held in Copenhagen. Dr. F. W. Hinkel of Buffalo reports a case of prophylactic use of serum preceding operation of adenocarcinoma of the nose in a patient with a history of repeated hemorrhages. The operation proceeded with an unusually small amount of bleeding. This case is quoted by Clowes and Busch.

RÉSUMÉ.

The evidence collected from the literature, as well as the experience from the three cases noted by the author, is predominatingly in favor of serotherapy for the control of hemorrhage, practically no failures having been reported when the serum was fresh and used in sufficient quantity in conditions indicating its employment. To the nose and throat specialist serotherapy presents a simple, efficient and harmless hemostatic agent—our salvation—in all hemorrhages which do not promptly come under the control of the usual methods, and to be used early before extreme loss of blood has occurred. The office and hospital equipment should always have a supply of precipitated blood sera on hand for emergency.

The fresh human blood serum is available to the author at all times through the courtesy of the staff of the New York Lying-In Hospital, to whom his thanks are due, as well as to Dr. ——————, through whose courtesy the author was able to present the first case.

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VIII.

THE SIMULATION OF PARANASAL SUPPURATIONS FOR TEACHING PURPOSES.*

By Greenfield Sluder, M. D.,

St. Louis.

Mr. President, I present to you a scheme for simulating the various suppurations of the accessory sinuses of the nose. So far as I am aware, such a one has not heretofore been used for teaching purposes. I submit to you the human adult cranium, which has been decalcified in ten per cent HCl (to facilitate dissection), the calvarium removed, the accessory sinuses opened by dissection from its cranial aspect (which in the decalcified specimen is easily done), and the lower jaw and tongue removed for reasons which will appear later. The specimen may not have been decalcified or may not have been hardened in alcohol or formaldehyd, according to your whim. It is then stained red in an effort to reproduce the color of the living tissues. This particular one was hardened in formaldehyd, and stained in ammoniacal solution with carmine, which I believe to be the best stain. Its subsequent preservation has been in a five per cent aqueous solution of benzoate of soda, which seems neither to soften nor harden the specimen that has once been thoroughly hardened by alcohol or formaldehyd. Should sodium benzoate be used as the primary preservative, it must be much stronger than five per cent solution, and even then does not answer the purpose at all well, because it does not harden it; but as a preservative for hardened macroscopic specimens it seems ideal, inasmuch as they do not mould or change by decomposition and may be handled in bare hands without corrosive effect on the skin. The jars should be kept covered. Uncovered jars develop a white mould should the solution be too weak. It does not, however, seem to injure the specimens. In warm temperatures this solution may slowly soften specimens that were not thoroughly hardened. Should this happen the specimen may be rehardened by a little formaldehyd. It is then washed and reimmersed in benzoate solution.

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*Read before the American Laryngological Association, May 25th, 1915.

The simulation of pus, a sample of which I submit for your observation, is the subcarbonat or hydroxid of bismuth held in suspension by the presence of mannite, an hexatomic alcohol. Nitrat of bismuth is dissolved in dilute nitric acid and about two per cent mannite added. Sodium carbonat or hydroxid solution is added to neutralization. The precipitate is washed and allowed to settle, or is centrifuged to the thickness of buttermilk. I have stained it by the addition of a slight amount of anilin yellow and methylen blue. This preparation may be diluted with distilled water to reproduce the seropurulent discharges, or may be centrifuged to reproduce the thicker purulent discharges. A drop of it is then introduced into a sinus which has been dissected open from its cranial aspect. It may be put into the sinus to overflowing, or into the normal outlet of the one that the experimenter may wish to demonstrate. The specimen is then observed as though it were the head of a patient in the erect posture, by means of head reflector, nasal speculum and postnasal mirror. The tongue and lower jaw being removed, makes this by far the easiest type for demonstration. Should the demonstrator desire to simulate in further verisimilitude, reproducing some of the difficulties that beset the examination of the living, the lower jaw and tongue may be left upon the specimen, the mouth pried open by a gag, and a tongue depressor put into service. The specimen is observed for the entrance of the drop which has been introduced from above. One may see it appear for the waiting, and my experienec is that the simulation of the clinical picture hereby produced is so true to life as to be astounding, and all the subtle variations may be reproduced by quantity of the "pus" mixture introduced, or its dilution. (Should the bismuth mixture dry in position during the course of the demonstration, it may be dissolved by immersion in ten per cent HCl; or it may be left in its position as a permanent demonstration.

This sterile and permanent preparation stained to simulate pus may be utilized upon the living in the clinic for more advanced students by the simple introduction of a drop or two through a slender cannula at the various points at which appear clinically the sinus suppurations.

IX.

CLOSURE OF POSTAURAL OPENING FOLLOWING SCHWARTZE-STACKE OPERATION, BY A MODIFICATION OF THE MOSETIG-MOORHOF METHOD.

By Hugh B. Blackwell, M. D.,

New York City.

The operation does not quite resemble the Mosetig-Moorhof operation. Dench describes a somewhat similar operation in his book on Diseases of the Ear, in his account of the Mosetig-Moorhof method. In the Mosetig-Moorhof operation a flap is mapped out, beginning two millimeters below the retroaural opening, slightly larger than the opening to be closed (Figure 2). This incision is carried through to the periosteum, and the flap so formed elevated from it; the edges of the postaural opening are now dissected back for a distance of two millimeters (Figure 1) and are sutured to the edges of our flap (Figure 3), which we turn upward into the postaural opening of the cavity, and the raw surface of the flap is directed outward. This raw surface is allowed to heal by granulation, or is subsequently grafted. The skin is now brought together over the denuded area from which the flap was taken (Figure 4).

In this particular case the flap was cut as in a Mosetig-Moorhof operation (Figure 6), but the edges of the opening to be closed, instead of being dissected back for two millimeters, were dissected back for three-eighths of an inch (Figure 5). The flap was turned upward into this opening, its skin surface directed inward, and sutured by one suture at the top opening (Figure 7). The edges of the opening were then brought together over the flap, bringing the two raw surfaces together (Figure 9). The skin was now brought together over the surface from which the flap was taken (Figures 8 and 10) as in the Mosetig-Moorhof operation.

The essential point of difference between the two operations is that at the conclusion of the Mosetig-Moorhof operation the raw surface of the flap is left uncovered, directed outward (Figure 3), and allowed to heal by granulation. In the case here described, the external raw surface of the flap was covered over by the skin of the surrounding tissues (Figures 7 and 9), the healing being more rapid, with a firmer resulting closure. At the conclusion of the operation the Schwartze cavity was packed with plain sterile gauze, per canal, care being taken not to roll up the skin edges of the inverted flap. An external dry dressing with bandage was then applied. The pressure exerted by the internal packing against the external dressing serves to hold the raw surfaces of the flaps in firm apposition.

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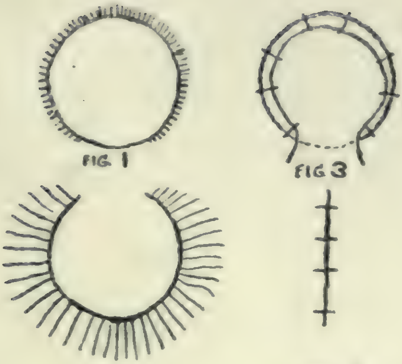

FIG. 1 FIG. 3 FIG. 2 FIG. 4

Mosetig-Moorhof Operation.

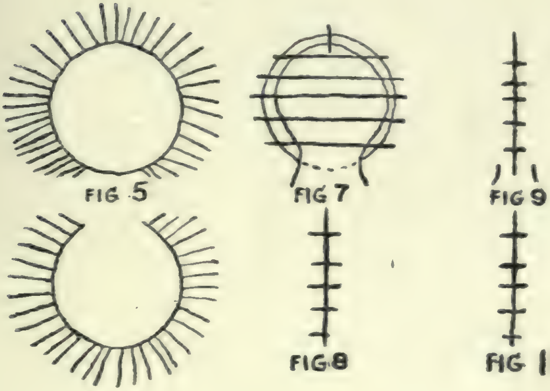

FIG. 5 FIG. 7 FIG. 9 FIG. 6 FIG. 8 FIG. 10

Modification of the Mosetig-Moorhof Operation.

The lines radiating from the margins of Figures 1, 2, 5 and 6 are intended to represent the extent of elevation of skin from the underlying tissues.

SOCIETY PROCEEDINGS.

PROCEEDINGS OF THE

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY.

May, 1915.

Technic of Suspension Laryngoscopy, With Lantern Slides and New Instruments.

Dr. Robert Clyde Lynch presented his modification of the suspension apparatus of Killian, with the latest improvements which he has devised to overcome difficulties encountered with the older models. He described the technic as now employed by him. The chief modifications in the apparatus concern the traveling crane, the limited motion of which is overcome by his table top; the Killian hook, which he has so modified as to greatly increase its feasibility; the spatula, which was made of tool steel, so that it is perfectly rigid under the greatest strain.

With the table top with crane attached, arranged so that the base of the crane will be about four inches in advance of the shoulders, with a suction apparatus at hand to care for the secretions, and with the patient quiet and relaxed by a proper anesthesia—in other words, with the patient properly suspended—the operator has both hands perfectly free for the most accurate work upon the larynx. Instead of pulling, or pinching, or biting at the tumor mass, one can pick it out accurately and carefully . The laryngologist, with this method, can work in the larynx as the general surgeon works upon the skin, or the gynecologist works in the vagina. The gagging or other motion of the patient is practically impossible, for the larynx is immovably fixed.

DISCUSSION.

Dr. Robert Levy, Denver, complimented Dr. Lynch upon his work. Few laryngologists had the ability to apply suspension laryngoscopy as Dr. Lynch had done, or the skill to devise methods for the improvement of the original technic, which was very unsatisfactory. He had been particularly interested in the tongue depressor, because at first this had been one of the difficulties, and to keep the tongue out of the way was one of the problems. The object of the original tongue spatula was to keep the tongue in the median line, to press it out of place, and at the same time to prevent the overlapping of the margins of the tongue. Dr. Lynch's instruments had entirely met these requirements.

Dr. Wolff Freudenthal, New York City, mentioned two cases that had come under his observation during the last two weeks which demonstrated the necessity for some form of suspension apparatus. The first patient had been sent to him from out of town. The man was suffering from tuberculosis of the right lung, was very much run down, and could not swallow at all. His physician wrote that unless the epiglottis, which was very much inflamed, could be removed, the end was near. The man's pulse was so weak that when he was carried to the operating room it was necessary to stimulate him for half an hour. Local anesthesia was employed, he was put under suspension, and the operation performed in the shortest time possible. The whole process healed within two weeks, the patient returned to the mountains, and was able to swallow food. His ultimate recovery, of course, was uncertain. The second case was that of a woman who had swallowed a fish bone. She went to a clinic near by, and the young intern sent her to the operating room, but failed to locate the foreign body, thought it was out, and dismissed the patient. Ten days later she consulted the speaker, at which time the entire left side was swollen, especially in the region of the arytenoid. Thinking she had an abscess, he made an incision, but got no pus. He put her under suspension, under general anesthesia, and removed the entire mass. The foreign body was not found, but the infiltration was extensive. When she recovered from the narcosis she became cyanotic from the blood running down, but this was removed, with some clots, and she recovered quickly.

Dr. William B. Chamberlin, Cleveland, mentioned a case operated upon by him last winter, shortly after his return from a visit to Dr. Lynch in New Orleans. The case was reported in the January number of the *Laryngoscope*. The patient was a child eight months old, and had swallowed an open safety pin, which was removed without difficulty by the suspension method. He had had a second case in a child eleven months old, in which he had operated successfully by the same method. A third case was a baby two weeks old. The infant had just been removed from the maternity hospital, when the father came in and found the child choking. On looking into its mouth a large safety pin could be seen in the hypopharynx. When the speaker was called in he thought he was going to see a child two months old, and was discouraged when he learned it was only two weeks old. However, the pin, which was a very large one, was easily removed by turning under suspension. By any other method its removal would have been exceedingly difficult.

Dr. Arthur I. Weil, New Orleans, had seen a good deal of Dr. Lynch's work, and had done a good deal of suspension work independently. He could personally testify to the value of the innovations Dr. Lynch had introduced. He called special attention to the value of this table top with suspension. He had formerly employed suspension with the crane attached to the head of the table, but the work was not nearly so satisfactory as with this extension table top devised by Dr. Lynch. The examination was rendered very much more reliable by this means than where one had to rely upon the screw of the suspension apparatus itself.

Dr. John W. Murphy, Cincinnati, mentioned a case which he had expected Dr. Lynch to operate upon on a proposed visit to Cincinnati. The patient's condition, however, became so bad before Dr. Lynch arrived that it was necessary to give him relief. He was a boy twelve years of age, with a papillomatous growth which almost occluded the larynx. It was necessary to resort to tracheotomy and the removal of a sufficiently large portion of the papilloma to enable the boy to breathe. Several weeks later Dr. Lynch succeeded, by his method, in cleaning out the rest of the papilloma. It was interesting to note the number of onlookers who could see almost every manipulation which the operator made in this larynx. In a week's time this boy was up and able to return to his occupation as a jockey.

Dr. Lynch, in closing the discussion, referring to the question of anesthesia, said he preferred general anesthesia, with ether. Warmed ether vapor, as delivered by one of the new methods, had proved very satisfactory. On a recent visit to New York City he had had the pleasure of operating upon a patient under rectal anesthesia, administered by Dr. Gwathmey. He had had no other experience, but in this case the method worked admirably. There was complete muscular relaxation, the amount of mucus was less than usual, and the exhalation of ether from the lungs was hardly perceptible. He liked to work under cocain analgesia in patients who were not afraid. Morphin and scopolamin were given first, then a solution of twenty-five per cent cocain in fifty per cent alcohol was employed. One or two drops of this solution in the hypopharynx and on the laryngeal face of the tongue, then one or two drops of an aqueous solution of cocain into the larynx, had given good results in his experience, enabling him to work for an hour or an hour and a half without difficulty. The suction apparatus which he used was a dental vacuum apparatus on an ordinary turbine pump, with a long tube which could be run down into the larynx and trachea for the purpose of removing clots or blood or anything else. After the patient had been under suspension for twenty minutes or half an hour, thick, ropy mucus accumulated in the pharynx, and this should be removed before the patient was taken from the suspension apparatus; otherwise this mucus would drop down into the larynx and choke the patient. He had never had vomiting under suspension.

The Tonsil Operation—An Inquiry Into the Actual Results Observed in a Series of Cases.

Dr. D. J. Gibb Wishart: Not feeling comfortable in permitting the enucleation of the tonsil in every child where operation appears to be called for, and yet not having been able to glean anything from the abundant literature provided upon the question, which would show him the proper course to pursue, the author set out upon the examination of cases which had been operated upon since he adopted the enucleation method, ten years ago. He hoped thus to arrive at some data which would enable him to act with greater confidence, both with regard to his private clientele, and in the instruction of students. Letters were sent to each patient on the history cards, just as they came, and with no omissions or selections. All cases which presented themselves were tabulated. The results were noted under the following heads: (1) Occupation; (2) sex; (3) age at time of operation; (4) years since operation; (5) previous operations, if any; (6) symptoms complained of; (7) effect of operation upon these; (8) anatomic conditions resulting from operation; (9) effect of operation upon voice; (10) effect of operation on general well-being; (11) symptoms unrelieved; (12) remarks.

The records compiled were from: private patients, twenty-eight cases in all, of which five were obtained by correspondence, and twenty-three examined personally in his office; those obtained from the hospital clinic, by Dr. McKelvey, seventeen in number. Because of faulty histories or the stupidity of parents, the records in the latter division were incomplete.

Sex and Age.—Three to fifty-two years, the cases being about equally divided between the sexes up to fifteen; after puberty, in the proportion of two males to eleven females.

Time Since Operation.—Average, five to seven years; no instance less than two years; in seventeen cases, eight years and over.

Effect of Operation Upon Symptoms Complained Of.—Not only were the symptoms (tonsillitis, ten cases; frequent colds, fourteen cases; enlarged glands, three cases; voice defects, eleven cases; swelling and aching throat, twelve cases; ear affections, ten cases; rheumatic symptoms, four cases; and headache, cough, enuresis, neuralgia, iritis, debility, restlessness, chorea, endocarditis, quinsy, dullness, etc., in one or more instances) complained of relieved, but it was notable that in nineteen cases the health was decidedly improved, and in no case was it deteriorated. The results of operation upon the symptoms actually present at the time when consultation took place were so uniformly good that the operation itself was justified. It was to be noted that in hospital cases where portions of the tonsil remained in situ, the effect upon symptoms was no better, if as good, as in private patients where enucleation was complete. It was not apparent that leaving a portion of the tonsils resulted in any benefit whatever to the patient, nor did the complete removal exert any deleterious effect.

Anatomic Results.—In patients from three to eleven years of age the results were apparently perfect in seven cases, the position of the palate normal in eleven cases, and in three cases there were minor variations which might or might not be considered as defects. In one private case a small patch of tonsil in the lower left sinus gave rise, seven years after operation, to swollen glands. In six clinical cases there were defects, chiefly the presence of small tags. In cases from twelve to fifteen years of age, results were apparently perfect in two cases; in one there was a marked variation in the curve of the palate arch; in one there was obliteration of the pillars, and in three there were scars.

In cases from twenty-one to fifty-seven years of age the results were apparently perfect in five cases. In three there were minor variations, not necessarily to be considered as defects. In four cases there were real defects. In the most seriously deformed of these last four the voice was definitely improved, and in one a good singing voice had been developed. In the cases in which the tonsil was extirpated in its entirety the anatomic results were undoubtedly best. In ten cases the notes failed to show whether there were adventitious bands of adhesion due to repeated attacks of inflammation in the tonsils, but the lesions of the pillars found present after operation could undoubtedly be ascribed to such a condition. The patients who had suffered from repeated attacks of tonsillitis were those in whom scars and malpositions were most numerous.

The effect upon the voice was carefully inquired into in all cases. Careful analysis of the results led to the deduction that the effect of extracapsular enucleation upon the singing voice or fatigued voice was not prejudicial, but might be considered beneficial.

General Well-being.—Practically every case was singularly corroborative of the view, long held, that the removal of diseased tonsils is attended by marked improvement in the general health of the patient.

Symptoms Unrelieved.—In fifteen cases these were mentioned. In three the nose was the seat of the trouble; in five the symptom complained of was aside from the question. In two endocarditis were noted; once as a new symptom, once as recurring in a postoperative attack. In one case nervousness and choreic tendency were thought to be independent of the original throat condition.

Remarks.—Under this head the author directed special attention to certain features of individual cases. The results of his investigation are presented in tabular form.

Partial Paralysis of the Soft Palate Following Removal of Tonsils and Adenoids. Observations on the Tonsil and Adenoid Operation.

Dr. Dunbar Roy: The patient, in the case reported, was a male, four and a half years of age, operated upon in the hospital May 20, 1913, for removal of tonsils and adenoid tissue in the nasopharynx. Complete removal was accomplished without trouble and with less traumatism than usual. Two days later the patient was out playing, with no signs of bad after-effects. The throat was not painful on deglutition, and there was but a slight amount of exudate in the tonsillar cavities. Ten days after the operation the child was taken to the writer's office with the history of having, two days before, a temperature of 101°, some malaise and some inability to articulate distinctly. In attempting to swallow liquids some of the fluid came back through the nose. Two days before noticing these symptoms the child had been allowed to eat an excessive dinner.

Examination showed the pharynx and nasopharynx to be in good condition, with only slight signs of inflammation. The tonsils had been thoroughly extirpated, and both pillars appeared normal. There was no pain. The tongue was white and coated. When the patient talked there were all the signs of paresis of the soft palate, just as is frequently seen after diphtheria. The family physician expressed the opinion that the paresis was due to intestinal toxemia. Laxatives, strychnin, light diet and irrigation of the nasopharynx with alkalin solutions, followed by touching with a solution of argyrol, brought about prompt improvement in the local and general condition, and the child was soon apparently perfectly well, except for the voice and a leathery appearance of the soft palate. The patient continued to improve, but it was nearly two months after the operation before the voice was practically normal.

The ill effects upon the throat following the radical removal of the tonsils and adenoids have been commented upon by many observers, but the author had encountered no mention, in the literature of the last few years, of the sequela of partial paralysis of the soft palate. Inexperienced operators, obsessed with the idea of radicalism, have caused much damage to throats which would otherwise have been better had they never been touched. Operation for the removal of tonsils and adenoids should not be looked upon as a simple procedure, but should have the same thought as any other major surgical operation. The operator should find the one technic which gives him the best results—that which leaves the throat in as normal a condition as possible—and should adhere to this in all his operations.

In the case reported, the only one the author had ever seen, owing to the fact that tonsillectomy and adenectomy were both performed, it was impossible to say which operation was the cause of the paresis. On the other hand, it might be argued that the paresis was accidental and entirely independent of the operation, especially since cases of paresis of the soft palate had been reported as being one type of chorea. The fact that the paresis occurred ten days after operation did not entirely exclude the condition as standing in the light of a propter hoc. His own opinion was that the paresis followed the adenoid portion of the operation, and that infection was the result of a traumatism on the posterior surface of the soft palate, which was manifested by the thick leathery condition found ten days after operation.

In the light of this experience he was convinced that operators too frequently use an unnecessary amount of force and traumatism in the removal of adenoids, especially in very young subjects, in whom the nasopharynx is very small. In the case reported he used small sized Brandage forceps for the removal of the adenoids. Since that time he had abandoned the forceps in this operation, because of the danger, in opening the forceps in the nasopharynx of small children, of traumatism of the soft palate. The La Force adenectome, without the large anterior bulbous portion, followed by the use of small Gottstein curettes, was, to him, the ideal operation.

The author gives the replies to the inqury, "Have you ever seen a total or partial paralysis of the soft palate following the removal of the faucial tonsils, nasopharyngeal adenoids, or both?" which he directed to fifty members of the society.

DISCUSSION.

Dr. George L. Richards, Fall River, gave briefly a review of one hundred and eighty-eight cases in children operated upon by himself and his associates during the last two years. It was found that the result was good in one hundred and forty-four cases, or seventy-seven per cent. By good he meant that the anterior pillar was there as a folded-over pillar, and not as a narrow line. The result was fair in thirty-eight cases, or twenty per cent. By fair he meant that there remained some scars. There were six cases, or three per cent, in which the results were poor. The voice was very bad in two cases, in which the uvula was caught and in which there was a distinct scar. He thought these would have a nasal tone, but they sang the scale perfectly well. He had reports of some of the children covering two years. It was a curious fact that if the anterior pillar alone was injured and the posterior pillar was not, the child had a good voice. Of the one hundred and eighty-eight cases, twenty, or nine per cent, had scars. By scars he meant contractions in which a little line could be seen. There was a tendency on the part of nature to replace more or less lymphoid tissue, and this manifested itself in granulations in the pharynx. Many patients complained of this, which gave a "scrapy" sensation, especially in winter. There were forty-five cases, or twenty-five per cent, in which these granulations could be seen. The pillars were injured in twenty-three cases, or twelve per cent, some sort of a little nick or injury having been inflicted. It was very easy to leave some remnant of the tonsil; this had been done in sixteen cases, or eight per cent. There was no hemorrhage. The patients, operated upon in the morning, were kept in the hospital until afternoon and then allowed to go home. Whether complete mouth breathers or not, sixteen, or eight per cent, breathed through the mouth where a part of the adenoids had been left. In order to be absolutely accurate as to results with reference to the voice, one must make records of this before the operation. Five, or two per cent, of his cases already lisped.

Dr. Richards presented a plaster model of the pharynx, upon which he demonstrated the relationship of the tonsils and the pillars. After a perfectly satisfactory operation the anterior and posterior pillars should be distinct and not fused together.

Dr. William B. Chamberlin, Cleveland, considered any method of tonsillectomy good which removed the tonsil and nothing else. The structure most easily injured was the posterior pillar at the junction with the uvula. The posterior pillar, which was extremely delicate, could be torn, just as could a piece of cloth. A quadrilateral palate might result. In his experience, as doubtless in that of others, disappointing results had been obtained in cases in which he thought the operative procedure absolutely perfect. This was due to the contraction of the scar tissue. He had never had an unsatisfactory result so far as the voice was concerned, not even for its finer uses, as by singers and public speakers.

Sometimes, in the removal of the adenoids after the tonsil operation, laceration of the posterior pillar was noted. This was the result of the gagging of the patient, plus the traction of the curette, thus causing injury to the pillar. It was important, after the adenoid operation, to palpate the nasopharynx to see that every portion of adenoid tissue had been removed. Injury to the posterior pillar might result from rough handling in the examination by the finger. If, with the adenoid curette, one continued the sweep too far downward, it was easily possible to strip off the entire mucosa of the posterior pharyngeal wall, leaving cicatricial tissue instead of healthy mucosa.

DR. J. A. STUCKY, Lexington, considered this study of the results of the tonsil operation very important. In his section of the country tonsillectomy was being performed not only by general practitioners but by general surgeons. In his college town there were several thousand students, and many teachers were constantly referring pupils to him for some defect in speech and vocalization. The society should go on record as advocating that the tonsil operation is not a minor operation, and that it is underestimated and underpaid. It should be considered as a hospital operation. Those who teach students should see that every step of the operation is seen by them. The point made by Dr. Chamberlin was a very important one. It should be done gently, however. The fingers of many operators were too large for this, and for that reason he advoctaed the use of a postnasal sponge holder, with which one could go in and swab out the pharyngeal vault as carefully as the gynecologist would swab the fundus of the uterus.

DR. WENDELL C. PHILLIPS, New York City, referring to Dr. Wishart's statement concerning Case 12 of the series, that operation was probably done too late in life to be useful so far as hearing was concerned, said if seven years were added to the patient's age it would then be time for otosclerosis to appear; besides, the mother had deafness. Earlier operation would have had the same result. So much had been said from time to time about the danger of removing the tonsils from singers, that he had become very careful in this regard. He had not had an extensive experience in operating on singers, but in every case in which he had removed the tonsils, not only had the voice not been injured, but it had been improved by the procedure. Within the last few months he had advised the removal of the tonsils from a man, a singer, a patient of Dr. Babbitt's, who had typical cheesy masses in the crypts of the tonsils. After being told that if he were to read the literature of the subject he would be discouraged as to the possible effect of the operation upon his voice, the patient said the effect of the tonsillitis on his voice was so bad that he might as well have the operation, as he would not sing much otherwise. Dr. Babbitt removed the tonsils and the man's voice was very much improved.

Dr. John F. Barnhill, Indianapolis, said it should be regarded as a settled fact that tonsils should be removed completely. He had been particularly impressed by Dr. Wishart's statement to the effect that imperfect operations were done abroad; that he had seen many of these cases, done in America, and had had to do the work over. The speaker had never regretted taking out tonsils completely whenever it was necessary to take them out at all, and he had always regretted imperfect work. He thought some of the symptoms mentioned following tonsillectomy were not due to the tonsils at all, but to some of the structures not removed. For instance, if any diseased portion of Waldeyer's ring were not removed, it would continue to become inflamed. The adenoid ring in its effects might be confused with the old trouble. The lingual tonsil became inflamed over and over again. There was an attempt on the part of nature to restore some of the tissue which had been removed, and there might be soreness of the lingual tonsil which did not exist or was not noticed before. He had seen paralysis of the soft palate following these operatoins, as suggested by Dr. Roy, and had considered it due, in some cases, to infiltration, and in some to traumatism caused by the finger in the vault of the pharynx, together with marked contraction of the soft palate. He had seen sloughing of the uvula in some of these cases. He cited one case in which the uvula, which had apparently not been injured, sloughed away ten days later. No harm resulted from it.

Dr. Sidney Yankauer, New York City, agreed with the other speakers with reference to the importance of avoiding roughness in handling the tissues in tonsil and adenoid operations. Undoubtedly many of the bad results reported from time to time were due to this error of technic. It was not difficult to imagine the amount of injury that might be done by an inexperienced operator with a rough gauze sponge rubbed about in the pharynx. For a number of years he had used a suction apparatus for the removal of blood and mucus from the throat, thus eliminating all traumatism. He had discarded gauze sponges entirely; whenever it was necessary to use any sponges, he employed soft sea sponges. In this way he had been able to avoid all traumatism, and the patient could take a full meal in from twenty-four to forty-eight hours.

Dr. James F. McCaw, Watertown, cited a case called to mind by Dr. Roy's paper. The patient, a little girl, five years of age, had paralysis of the soft palate immediately following the adenoid and tonsil operation. He had attributed it to his overzealousness in attempting to rid the child of every vestige of adenoid tissue by using an overlarge adenotome. The paralysis lasted seven or eight months.

Dr. G. Hudson Makuen, Philadelphia, wished to say a word in his own defense, inasmuch as reference had been made to something he had written bearing on this subject. One thing upon which he had ceased to speculate concerning the tonsil operation was the idea that the mere removal of the faucial tonsil injures the voice. When the removal of the faucial tonsil was absolutely indicated, and when it was properly removed, no injury could result. When its removal was indicated, the tonsil itself was a menace to the voice. The thing that injured the voice was the faulty removal of the faucial tonsil or the adenoids. An operation which tended to paralyze or to mutilate the soft palate, must of necessity injure the voice. An important thing in this connection was what might be called the musical instinct. If a singer had a musical ear and a musical brain, he would sing fairly well even with a damaged pharynx. Just as good violinist could make good music on a poor instrument, so a good singer could have a good voice with an indifferent vocal instrument.

Dr. George F. Keiper, Lafayette, had two cases of paralysis of the soft palate following the tonsil and adenoid operation, due, he thought, to the use of the palate retractor—or protractor, as this instrument should be called. If in inserting the finger one would hug the posterior pharyngeal wall, he would not be apt to injure the soft palate, even if the patient gagged. He had abandoned the use of the retractor.

Dr. Clifton M. Miller, Richmond, emphasized the importance of warning patients who sing not to begin to use the voice in the higher registers too soon after the tonsil and adenoid operation. He cited the case of a young lady whose tonsils he removed, warning her not to use the voice in the higher registers for six weeks, but to confine her use to the middle or normal registers. At the end of three weeks she felt so well that she began to sing in the high registers, and her voice broke. The mental effect was so profound that instead of being able to use the upper registers in six weeks, it was six months before she could do so. The psychic effect on these patients, with their highly organized nervous systems, was apt to be very profound, and for this reason they should be emphatically warned.

Dr. Greenfield Sluder, St. Louis, emphasized the point made by Dr. Richards in reference to nature's attempts to reproduce lymphoid tissue after tonsillectomy. He called attention to the fact that the lingual tonsil gives rise to a lymphoid growth which extends into the tonsillar fossa and at times reproduces what is anatomically perfectly corresponding to the original tonsil and clinically takes on all of its pernicious activities. He mentioned also that in many cases pharyngitis lateralis was developed, also being at times clinically pernicious. Whether these reproductions of lymphoid masses be a part of nature's effort in healing, or whether they be a manifestation of continued diseased activities, is a question that he has not been able to solve. In the *Archiv. für Laryngologie*, for 1912, Dr. Oswald Levinstein described what he termed a new pathologic tonsil which developed in the lateral aspect of the tongue, just anterior to the palatoglossus fold. I have confirmed Dr. Levinstein's observations, finding this hypertrophy of lymphoid follicles massed together in such a way as to resemble a tonsil in cases of "painful tongue," the condition described by Sir Henry Butlin in his monograph, "Diseases of the Tongue," in which the lymphoid follicles at the junction of the palatoglossal fold with the tongue proper were the only demonstrative lesions. Lëvinstein attributes this tonsillar development to irritation in the lymphoid tissue. This surely seems to be a development in a pathologic line.

He had also been interested in the question of the production of scars in the throat, and had frequently noted the quadrilateral palate mentioned by Dr. Chamberlin. This might occur, regardless of the method employed for the tonsillectomy, and comes to pass because of the fact that the operator cannot control the fixing or focusing of the scar which must needs develop. He thought it advantageous to focus the scar anterior and below to draw the palate downward and forward, which pulls it away from the eustachian tube above. He thought this to be a definite help in cases where the tonsil is removed because of the eustachian tube irritation. He did not believe, from an extensive observation, that this interfered in any way, shape or form with speech or the artistic use of the voice. He mentioned that he had dwelt upon this point (drawing the palate downward and forward) in his paper to the American Medical Association, in 1910, in which he described a "Method of Tonsillectomy by Means of the Alveolar Eminence of the Mandible and the Guillotine."

Answering a question by Dr. Richards regarding his earlier cases, Dr. Sluder explained that he had formerly considered the plica tonsillaris as a part of the pillar. It was with this idea in mind that he made the statement that he always removed a portion of the anterior pillar, which tended to fix or focus the scar downward and forward, and that usually a few muscle fibers were attached to it. The careful examination of hundreds of tonsils reveals that very seldom is much muscle fiber attached, and that with a little attention to this point the entire palletoglossus may be pushed aside and the plica, with the membrane covering it, removed, in no sense and in no degree injuring the pillar proper.

His ideas concerning the pillar and the plica are gained from the work of Dr. George Fetterolf on the "Anatomy of the Tonsil and Its Capsule," published in the *American Journal of Medical Sciences,* in 1912. Fetterolf's conception is that the plica tonsillaris is a fold of the capsule of the tonsil pushed inward over the tonsillar mass, carrying a layer of mucous membrane with it, and that the pillar proper is constituted by the palletoglossus muscle. In other words, the statement made in the speaker's paper of 1910 was a confusion in the nomenclature.

Dr. Wishart, in closing the discussion, said he had not met with any continued paralysis of the soft palate after operation, and he had used the finger very largely; never, however, while the patient was retching. He always saw to it that the patient was in the proper condition of anesthesia before he attempted to remove adenoids. He would rather give a little more anesthetic than to run the risk of overstretching the soft palate. Many of the bad after-effects were caused by too much sponging. The assistant should stand in the right position with referenec to the patient, so that he could see the parts, and the sponge should never be turned round. He had merely cited Dr. Makuen's article and had presented these cases as notes upon it, not as a criticism.

Dr. Roy, in closing the discussion, said it had seemed to him important to bring the subject before the society, inasmuch as he had never heard of anyone, in the literature or otherwise, who had encountered a case of this kind, and that in writing to members of the profession he had found it much more common than he had imagined. He did not want to go into the matter of technic, or to advocate this or that method; he did say, however that one should not change from a well recognized technic, which one had used and knew how to use successfully, to a new method. In the majority of cases the operation was performed too hurriedly, often without stopping or waiting for the oozing, which frequently occurred, to cease, and without seeing just where the instrument was going. The correction of these errors in technic would lessen the number of traumatic and unsatisfactory after-results. He had never employed a suction apparatus, which, it seemed to him, sucked the blood out of the arterioles and veins and tended to keep up the bleeding. He operated slowly in doing tonsillectomy, and had very few drops of blood. A sponge put into the cavity and allowed to remain for a while would prevent bleeding. If, as Dr. Stucky had suggested, the operation were considered a major operation, always to be performed in a hospital, and if the operator were always careful to see the entire field and to know just where his instrument was going, better results would be obtained than had been previously accomplished.

Some Investigations of the Salivary Glands in Health and Disease.

Dr. Thomas E. Carmody: There is notable lack of literature on this subject, due to the rarity with which the salivary glands are affected, or to the fact that cases are not considered by observers as of sufficient interest to be reported.

Among pathologic conditions mentioned by the author as involving the salivary glands or ducts were mentioned: Calculus; injury to or stoppage of ducts by mucous plugs, constriction, or cicatricial bands; Miculicz's disease; tuberculosis; air tumors; new growths; chronic hypertrophy; atrophy; and congenital defects.

Mistakes in diagnosis of these conditions seemed to be the rule, probably due to the infrequency of most of them.

Was there any reason why the salivary glands are so free from disease? It had been suggested that these structures are more frequently affected in persons who are careless about their teeth, though this view could not be substantiated. A badly decayed tooth, however, was frequently found opposite the opening of Steno's duct in cases of disease of this duct or of the parotid gland. The rich blood supply of the tissues about the mouth could not explain the special immunity of the salivary glands; nor could the good drainage which characterizes the parotid, especially the superior portion, account for this relative immunity, because the submaxillary and sublingual are not so advantageously situated.

Although a good deal of work had been done to determine the protective action of the sulfocyanate of potassium, nothing had been definitely determined in this regard.

The continuous flow of secretion from the glands probably had as much to do as anything in preventing infection. But it must be remembered that in case of the kidney one would expect, on account of gravity, that there would be small chance of infection taking place by way of the ureter, and yet the kidney was probably more frequently involved than the salivary glands; in tuberculosis, for example, the kidney is affected twenty-five times to one of the salivary group.

The thought presented itself that the glands and tissues might be among the last to lose their vitality, a view which the author believed to be true. It had been his observation, from a limited investigation with glands obtained postmortem, that patients who were very ill with various diseases digested starch at ordinary room temperature and at forty degrees as well as the control of presumably normal saliva. The starch test, while the best at hand, left much to be desired. This test is made with a one per cent paste, made by heating the required amount of starch with a little water, and adding enough water to make a one per cent solution. Of this solution the tubes are taken containing five cubic centimeters each, and one drop of filtered saliva is added at intervals of one minute; one drop of a N/20 iodin solution is added and changes noted. The first few tubes show blue, gradually becoming lighter until, at the end of ten minutes, a straw color was given. The blue color was due to the reaction of the starch to iodin; the gradual change to reddish is for the reason that the starch is changed to erythrodextrin, which gives a red color; and on further digestion, achrodextrin is found, which gives no color. This, however, will not be found for at least twenty minutes at ordinary room temperature, unless a large amount of saliva is added. If tested at forty degrees the change takes place more rapidly. No quantitative tests for soluble sulfocyanate have as yet been made.

The author reported fifteen cases of disease and injury to the salivary glands.

Review of the Most Important Recent Contributions to the Treatment of Diseases of the Ear.

Dr. George L. Richards presented abstracts of literature on the following:

1. Closure of eustachian tube during the radical operation.
2. Radical mastoid operation.
3. Operative treatment of otitis media.
4. Meningitis.
5. Vaccines.
6. Blood cultures.
7. Use of radium.
8. Salvarsan.
9. Hearing power of the vestibule.
10. New instruments.

DISCUSSION.

DR. FRANK ALLPORT, Chicago, said the radical mastoid operation, which was a serious procedure, should not be undertaken without careful consideration. The spoken and written contributions to the subject during the past two years had tended to mitigate the enthusiasm of a good many mastoid performers, himself among the number. All other means should be tried before resorting to operation. Even in the most skillful hands the operation was not always successful. Nevertheless, he wished still to go on record as holding that, if something must be done, if all other measures of a conservative nature have been exhausted, if the point has been reached where some surgical procedure must be recommended, nothing had been proposed which would take the place of the properly performed, old fashioned, radical mastoid operation. The conservative method, as advocated by Heath and others, had very few enthusiastic followers, especially in this country. He had been surprised to read, in a paper by Haskin, the statement, which Dr. Richards quoted, to the effect that the author had not performed a radical mastoid operation on a private patient for some time. He could not understand how a man who sees a great variety of cases could arrive at this conclusion. In dispensary practice, where patients could not be given the care that private patients receive, it was necessary to perform the radical mastoid operation, according to Dr. Haskin, who claimed that by means of his vacuum cleaner he could obviate the necessity of doing the radical mastoid operation. There seemed to be some divergence of opinion as to what should be done with the eustachian tube in the radical mastoid operation. So far as he was concerned, there seemed to be but one way, and he paid a good deal of attention to the closing of the tube. Kyle, of Los Angeles, in a recent article, advocated paying no attention to the eustachian tube. A case might be perfectly well in every other respect, but if there is a constant dripping from the eustachian tube, it was certainly not cured so far as the patient was concerned. If the eustachian tube was left untouched, the patient would still go through life with a discharge. He thought, therefore, that strict attention should be paid to the closure of the mouth of the tube, by the Yankauer or any other efficient method.

DR. THOMAS J. GALLAGHER, Denver, considered Dr. Yankauer's contribution to the treatment of the eustachian tube, both in chronic suppuration and in stenosis, of great value. In the radical mastoid he had found this method very satsifactory. In many cases of chronic suppuration the closure of the tube would prevent a mastoid operation. However, it was not intended to take the place of the mastoid operation when the indications were clear for such procedure. He had found Yankauer's method extremely gratifying in stenosis of the tube, using, of course, the special instruments devised by him. In routine work the caliber of the tube should be known, and he had long since discarded the Toynbe tube with inflation to ascertain this fact. Even in cases of combined internal ear disease and tubal obstruction the tube should be made as patent as possible, which often improved the hearing, although the internal ear condition remained uninfluenced. In cases of chronic perforation often this could be closed if proper drainage is established.

Dr. E. B. Dench, New York City, referring to the question of bacteremia in sinus thrombosis, called attention to the point, not mentioned by the essayist, that in a certain stage of sinus thrombosis one should not expect to find bacteremia. In the development of this condition there was first the formation of the parietal clot. At first there was bacteremia; suddenly there was none, but the temperature continued. That meant that the clot formation was continuing. After two or three days the blood was again filled with bacteria; that meant that the clot was breaking down. He cited a case in which the sinus was accidentally wounded. He expected to find bacteremia, and had blood cultures made from the second day. At first there was no bacteremia, but after eight days there was. It was not until the clot broke down that the bacteria were found in the blood. In closing the eustachian tube he had found it advantageous, after curetting, to so place the skin graft that it laps over the anterior wall of the canal, then to force the graft down into the tube. When the tube was left open, instead of having dermatization of the tympanic cavity, there was proliferation of mucous membrane. Another plan which he had tried last winter was the use of a piece of bone shaped with the rongeur forceps.

At the request of Dr. Richards, Dr. Dench demonstrated the method of skin grafting which he had presented before the American Otological Society at its recent meeting.

Dr. J. A. Stucky, Lexington, emphasized the importance of a careful examination of the ear in every case in which salvarsan is to be administered. In his two cases, to which Dr. Richards referred, complete deafness in the ear previously affected with chronic suppuration followed the use of salvarsan. He had had more trouble with the eustachian tube than with any other part of the radical mastoid operation. He had had unsatisfactory results unless he managed to close the tube. The method he employed was that described by Dr. Dench, plus the use of the Yankauer curette.

Dr. Wendell C. Phillips, New York City, thought a paper like that of Dr. Richards' important, especially as it gave an opportunity for clearing up the records. It emphasized, too, the importance of being careful in making statements, an illustration of which called him to his feet. The members of the society must give Dr. Haskin credit for some very valuable work with reference to the vaccins, and for his radical mastoid work. It was unfortunate, however, that Dr. Haskin should have made the statement, in the contribution to which Dr. Allport referred, that he had not done a radical mastoid operation on a private patient in four years. That statement should have been qualified by mentioning the fact that he had had no cases of chronic middle ear suppuration with symptoms of meningeal or labyrinthine complications. The speaker did not believe Dr. Haskin meant to convey the idea that he would not do a radical mastoid operation in such cases. In the presence of distinct evidence of extensive cholesteatoma, no other treatment than the radical mastoid operation was indicated. These were the dangerous cases. As he looked back over his cases for the last few years, he could not recall a single case of fistula where he did not find cholesteatoma. Such cases could be classed as suitable for no other treatment than radical mastoid operation.

Dr. Richards, in closing the discussion, said if those who thought they would get rid of cholesteatoma, when present, in every case of radical mastoid operation, would study the pathologic anatomy of cholesteatoma of the temporal bone, they would be convinced that in many cases the cholesteatoma had penetrated many cells of the bone which could not be removed without destroying the life of the bone or of the patient.

Brain Abscess of Otitic Origin with the Report of Five Cases.

Dr. John W. Murphy: Five cases of brain infection of otitic origin are presented as illustrating that every case of suppurating ear is a menace to the patient. These cases are interesting (1) from the slight symptoms presented up to within a few hours of the time of operation; (2) in every case it was possible to trace the origin of the infection to a lesion in the middle ear; in three of the operated cases, at the time of operation, and in the two nonoperative fatal cases at a subsequent postmortem. The portion of the brain most likely to be involved from middle ear infection depends upon the manner in which the infection occurs. In the author's experience, when infection occurs by direct continuity, the temporosphenoidal lobe is most often the site of abscess formation. When the extension is through the labyrinth, then the abscess is most often on the anterior aspect of the cerebellum, since the abscess is most often found adjacent to the infected bone area.

In the first case, male, sixty-three years, developed soreness in neck and back of right ear following exposure to severe cold. No discharge from ear, but evidence of old chronic inflammatory middle ear trouble. Temperature 102° F. next day, with considerable pain along tendons of sternocleidomastoid muscle, with some tenderness at the tip. Mastoid operation failed to reveal the seat of the trouble until the entire tip had been explored. In the under surface of the tip there was a perforation about the size of a lead pencil, which opened into a large abscess at the tip. The pus had traveled along the tendons of the sternocleoidomastoid muscle, and had produced a typical Bezold's abscess, containing several ounces of thick, creamy pus. The entire pneumatic process was one mass of necrosed cells and pus. A large opening in the tegmen tympani communicated directly with the dura, through which a quantity of extradural pus escaped when the opening was enlarged and the dura depressed. The bone destruction was quite extensive. The middle ear presented no symptoms of the process, which was explained by the presence of firm granulations in the attic completely shutting off communication with the middle ear. Satisfactory recovery after radical mastoid operation and drainage of the Bezold abscess.

Case 2.—Male, aged fifteen years. Following exposure he complained of pain in the left ear, with beginning discharge the next day, and cessation of pain. Under treatment with hot boracic acid douches the discharge gradually lessened, and in about ten days ceased. Starting up again, a week later, despite free myringotomy by the family physician, the discharge continued. Symptoms simulated violent frontal sinuitis, yet examination was negative. Following a severe convulsion and unconsciousness, the author was consulted. The patient was profoundly unconscious. The left external canal was full of mucus-like pus.. From the symptoms, the diagnosis was made of localized acute meningitis. Opening and drainage of mastoid advised, but patient died, without having regained consciousness, early next morning. Postmortem examination showed the petrous portion of the left temporal bone much worse than the right, with several areas of roughened bone, showing the point of meningeal infection. The entire petrous portion of the bone was filled with pus and broken down bone cells. The left frontal sinus, the seat of the pain, was found to be normal. The tegmen tympani was also eroded. The left temporal lobe was much discolored, but no abscess was present. A well-defined acute meningitis of this lobe was present, which accounted for the intracranial symptoms, expulsive (?) obstinate constipation, Jacksonian epileptic seizures, and paralysis of the third nerve, all of which characterized the case.

Case 3.—Male, six years of age. Symptoms of mastoid involvement following a discharging ear. Simple mastoid operation, two weeks before the writer saw the patient, was followed by symptoms of cerebral complications. A diagnosis of temporosphenoidal abscess was made, and operation instituted. An enormously enlarged encysted abscess was found in the temporosphenoidal lobe. Complete recovery. Child well one year later.

Case 4.—Male, aged seven years. Operated upon by another surgeon for abscess of the right ear. Wound back of ear did not heal, but continued to discharge foul-smelling pus, varying in degree. Child seemed normal otherwise, and was in school. No cerebral symptoms. External ear dry, drum and hearing apparently normal. Thinking he had simply a sinus to deal with, the essayist opened the mastoid wound, taking his usual precaution, where he did not perform the mastoid operation, of going some distance outside the original incision, to lessen the danger of injuring dura or sinus, if either have been exposed.

He found that neither the antrum nor a single mastoid cell had been opened, but that a button of bone about the size of a silver half-dollar had been removed from the temporal bone. Through this opening there was a tense, bulging, nonpulsating dural protrusion. Exploration revealed an encysted temporosphenoidal abscess, from which was evacuated about four ounces of most offensive pus, in which there seemed to be much gas. The symptoms from which the patient had suffered for months were undoubtedly due to this encysted abscess, which leaked externally when the pressure became too great. This leakage doubtless saved the patient's life. Rapid convalescence followed evacuation of the abscess.

Case 5.—Male, aged thirty-five, was brought into the hospital in a profoundly comatose condition, and no history was obtainable. Pupils contracted, not responsive to light; corneal reflex abolished. Temperature, 99°; pulse, 190; respirations stertorous. Urine negative. Over right mastoid there was an old scar of a former mastoid operation. There was pus in the right external auditory canal. The patient died before he could be operated upon. Immediate examination revealed a large temporosphenoidal abscess. From the amount of cerebral fluid that came away with the pus, the writer was satisfied that the sudden torpor was due to the abscess bursting into the lateral ventricle of that side.

In these five cases, seen within a year, it was possible to demonstrate positively that a suppurating middle ear was the direct cause of the brain infection.

DISCUSSION.

Dr. Norval H. Pierce, Chicago, took issue with Dr. Eagleton in the matter of operating through a large opening in both skin and bone in infected areas. He had witnessed bad results from extension to the brain. He had never seen any reason for these large bone flaps in operating upon the brain. He had always attempted, in both cerebral and cerebellar abscesses, to operate through a small incision. He classified brain abscesses as those which occasionally recover, and those which never recover. The abscesses which occasionally recovered were those in which a socalled stalk of the abscess could be followed—in which this fistulous, necrotic pathway of infection could be followed up to the brain cavity. Perhaps the majority of such cases would recover. In transportation abscesses, where the infected material was transported through the veins or otherwise to the brain from the suppurating focus in the mastoid, where no pathway could be found, and where one blindly stabbed into the brain, failure to recover was the inevitable rule. One either missed the abscess or, even if it were found, there was such an encephalitis or meningitis from going through or infecting the arachnoid spaces, that recovery was impossible. It was difficult to thoroughly explore the wound in the mastoid in those abscesses situated at a distance from the original area of infection. If, therefore, he could not follow the stalk into the abscess, he took away a button of bone. He never laid back a flap. He thought it a very bad practice to explore through an infected area. It was a good plan to endeavor to form a plastic inflammation, before going through with an exploratory instrument, by packing the subdural space.

Dr. Wendell C. Phillips, New York City, congratulated Dr. Murphy upon having the privilege of treating so many cases of brain abscess in so short a time, also upon his large percentage of recoveries. Most of the symptoms, or socalled symptoms, of brain abscess were not symptoms of the abscess itself, but were symptoms which came on later, or after the general meningeal symptoms had appeared. Unfortunately, cases of encysted brain abscess presented very few symptoms until rupture of the abscess walls had taken place. Unilateral headache was the one symptom he had always found to be present. He had become suspicious of a severe and persistent unilateral headache in cases of chronic middle ear suppuration. In many cases it was not severe enough for one to obtain a definite clinical history. He had had a few cases that had impressed him so forcibly that he had come to look upon this type of headache as a symptom of brain abscess. One of these was a young and otherwise healthy student, who weighed one hundred and eighty pounds. He had chronic suppuration of the middle ear, with an excessive discharge. This discharge was so excessive and so foul that the patient wanted some kind of an operation for it. He did the radical mastoid operation without eliciting the symptom of headache. At the time of operation it was found that there was a dehiscence in the attic, and a few drops of pus oozed from a pin-hole opening in the dura. A large encapsulated abscess was found. On questioning the patient later it was learned that whenever he studied very hard he had severe headache on that side. Another case was one of cholesteatoma with fistula of the horizontal canal. That man complained of incessant headache, but had no other symptom. He was seen by several confrères, including neurologists, but no other symptom could be elicited. Later, operation revealed brain abscess. He asked Dr. Murphy what he understood by "large quantity" of pus.

Dr. John F. Barnhill, Indianapolis, agreed with Dr. Phillips. There were undoubtedly cases in which it was impossible to make a diagnosis of brain abscess, and these were cases in which there had been suppurative otitis media. The unilateral headache was not present all the time, but appeared some time during the twenty-four hours. Headache was a symptom of nearly every class of brain disease, especially of brain tumor; and it was very difficult to differentiate between brain abscess and brain tumor in which otorrhea had ceased. There were also the mixed symptom cases, in which there was considerable localized meninigitis along with the symptoms of brain abscess. He had seen a case very recently in which the pulse was fifty-five, the temperature one hundred and four degrees, and in which the type of headache described by Dr. Phillips was present in all its intensity. There had been projectile vomiting—all the symptoms, in short, usually associated with brain abscess. He counted the father's pulse and found it only fifty-nine. Slow pulse was probably a family characteristic in this case. He agreed with Dr. Eagleton in the matter of turning down a large bone flap in order to make a more accurate diagnosis and more thorough evacuation.

Dr. Frank R. Spencer, Boulder, said nystagmus, in cases of temporosphenoidal abscess, might be absent. As in one of Dr. Murphy's cases, it might come before projectile vomiting, and it might change suddenly. It might occur with the quick component to the right, and then change suddenly to the left. This had been emphasized by Ruttin and Neumann. He recalled a case which he and Dr. Murphy had seen, four years ago, in Ruttin's clinic. The patient, a young woman, had nystagmus which changed character twice while the patient was under observation in the clinic. The suddenness with which the quick component changed from side to side was very characteristic of cerebellar abscess.

Dr. J. A. Stucky, Lexington, said the method of drainage which he had found most satisfactory was that of Sir Victor Horsley, by means of several strips of rubber tubing. Through this rubber tubing the abscess cavity could be irrigated. The use of gauze was conducive not only to the formation of granulations, but to the opening up of areas of infection. The tubing could be removed one piece at the time.

Dr. S. MacCuen Smith, Philadelphia, congratulated Dr. Murphy upon the facility with which he diagnosed and located the abscess formations. In fifty per cent of his own cases the abscesses were found postmortem. Brain localization was one of the most difficult and very often the most unsatisfactory subjects with which to deal, notwithstanding the recent advances in this line. He corroborated what Dr. Pierce had said with regard to making a large opening.

Dr. Arthur B. Duel, New York City, said his experience accorded with that of Dr. Pierce with regard to the classification of brain abscesses. Those cases in which he had found a "stalk" leading to the abscess cavity had invariably recovered. Where he had found a stalk of entrance he had done very little except to open up the bone freely to give vent through the naturally made pipe. Perhaps nine out of ten cases of brain abscess reported as cured had been of this type. The unsuccessful cases had been those in which he had been unable to find a "stalk" of entrance. In a number of instances he had located and drained the abscess, but the patient had died from meningitis or encephalitis by extension of the infection. This type of brain abscess would be more successfully treated when otologists learned to employ the technic suggested by Dr. Eagleton, of turning down a large flap and observing the niceties of surgical technic. He emphasized the importance of avoiding puncturing the vessels, as the hemorrhage which would thus take place into normal brain tissue would cause encephalitis or meningitis.

Dr. George W. MacKenzie, Philadelphia, gathered that in many of the reported cases the abscess appeared to develop within a week or ten days after the mastoid operation. In view of the four stages through which an abscess had to pass in its development, he thought this too short a time. Referring to the use of a small knife, such as Dr. Murphy had suggested, he said one might strike the abscess and yet not get pus.

He advocated a crucial incision in order to find the pus. It was enough, when the pus began to come faster and faster, to put on the artery clips and dilate the opening. He had known of cases successfully operated in which the patient lived three or four weeks and then died. It was not easy to put in the rubber tube and keep it in place, nor was it always easy to drain these cases as they should be drained. It had been suggested that a brain abscess should be treated as an abscess in any other part of the body; to do this it would be necessary to make a large opening and curette the abscess cavity very gently. This would shorten the duration of an abscess. It was not always easy to locate an abscess. He cited a case in which he had recently been mistaken with reference to a temporosphenoidal abscess. The case in question was one that developed in the coures of a labyrinth suppuration. Besides the general symptoms common to all brain abscess cases, the focalizing symptoms seemed to point to the cerebellum. Upon incising the cerebellum rather freely no pus could be found. It was then decided to open the temporosphenoidal lobe, when a large abscess was found and drained. An interesting finding in the case was the presence of a horizontal, wide excursioned nystagmus to the side operated that was not present before the operation. In fact, prior to the operation the patient had a short excursioned rotary nystagmus to the same side. This horizontal nystagmus following the operation must have resulted from the incision made in the cerebellum, and corroborates the claim of Alexander and the speaker that irritation in the cerebellum produces horizontal wide excursion nystagmus to the same side, in contradistinction to the rotary short excursion nystagmus found in labyrinthine affections.

Dr. E. B. Dench, New York City, thought a bone flap absolutely contraindicated. A brain abscess must be explored through the stalk; failure to find anything could then be followed by making a flap. An ordinary curved director was better than a knife. He had devised a director in quarter inches, which could be carefully introduced until a free flow of pus was obtained. He then inserted a cigarette drain of gauze, which could be left in place for twenty-four or forty-eight hours, and which would give all the drainage needed. This could be replaced, as the abscess cavity collapsed, with folded rubber tissue.

Dr. Smith added that drainage of a brain abscess should be through the avenue of infection. All his cases which had recovered had been drained in that way, whereas cases which he had evacuated through decompression had uniformly died.

Dr. Murphy, in closing the discussion, said he had seen two infections of the middle ear with temporosphenoidal abscess. He had brought up this subject in order to emphasize the belief that running ears were more often the cause of brain abscess than had been thought.

A Report of a Collective Investigation on the Curettement of the Eustachian Tube in Chronic Aural Suppuration.

Dr. Sidney Yankauer published in *The Laryngoscope* of July, 1910, a procedure for the cure of chronic otorrhea. In order to ascertain what the experience of other otologists had been with this operation, he wrote to over six thousand otologists in the United States and elsewhere. Among nearly two thousand replies, he found one hundred and nineteen operators who had performed the operation from one to fifty-three times, the total number of cases operated upon being seven hundred and thirty-five.

Inasmuch as the operation is based upon the principle that the closure of the eustachian tube is essential to the healing of the diseased ear, it naturally follows that in those cases in which organic atresia did not result from the operation, a cure could not be expected, and the statement that the closure of the tube will be followed by a cure can be true only of those cases in which the tube has actually been closed. Both sets of figures are given. Of the seven hundred and thirty-five cases, the tube was successfully closed, after one or more curettings, in six hundred and nine, or eighty-three per cent of the cases. The number of patients reported as cured is three hundred and seventy-nine, or 51.5 per cent of the total number, or sixty-two per cent of the number in which the tube was successfully closed. In other words, more than one-half of all cases of chronic suppuration of the middle ear have been cured by closing the eustachian tube through the intact external auditory meatus and without other surgical treatment.

In the author's experience, confirmed by observations extending over ten years, it might be stated as a dictum that whenever the eustachian tube is permanently closed by organic atresia, a perforation in the drum membrane will never close and cannot be made to close. Whenever, therefore, in any case it seems to be possible to restore the continuity of the drum membrane, after the suppuration has subsided, and so restore the ear to a perfectly normal condition, the eustachian tube must not be closed. In such cases the operation is contraindicated, as it is also in those cases in which it is intended to perform one of the socalled conservative radical operations, which are also supposed to result in the closure of the drum membrane. Furthermore, the procedure is adapted only for the cure of a chronic suppurative process, and should never be employed in the presence of an acute exacerbation. In like manner, the presence of intracranial complications demands the immediate exposure of the entire diseased area, and to delay such exposure until the results of tubal atresia can be known would be entirely unjustifiable; besides which, in the presence of intracranial complications, even if these have become latent, any operative procedure upon the ear, short of a radical exposure of the entire diseased area, might be dangerous, as is suggested by the single death which has followed this operation. The history of this case is detailed.

Inasmuch as the eustachian tube, in the class of cases under consideration, has permanently lost its function, its closure is harmless; and, inasmuch as it assumes a perverted or pathologic function, its patency is detrimental and its closure has become a therapeutic necessity.

Only two hundred and sixty-six cases, or thirty-six per cent, were closed after the first curetting in this series, while two or more curettings were needed in two hundred and forty-four cases. Hemorrhage, sufficiently profuse to be designated as severe, was reported in only twenty-six, or 3.6 per cent of the cases. The hearing was reported upon in five hundred and sixty-four cases. It was improved in two hundred and fifty-eight, or forty-six per cent of the cases; unimproved in two hundred and eighty-one, or fifty per cent of the cases; and in twenty-five cases, or four per cent, it was made worse. Hence the hearing was improved in ninety per cent of the cured cases. Tinnitus was reported in three hundred and sixty-five cases, in which it was lessened in one hundred and eighty-nine, unchanged in one hundred and fifty-nine, and made worse in seventeen cases.

His procedure may be carried out under local anesthesia and in dispensary practice.

This collective investigation justifies the following position: Whatever method is adopted to secure the healing of the diseased middle ear cavities, whether nature be allowed to effect the healing unaided, whether the efforts of nature be stimulated by the administration of vaccins, whether nature's work be made easier by the removal of some of the diseased parts by minor surgery, or whether her work be made easiest of all by radically removing all diseased tissue, in any event, in every case reinfection of the diseased area must be prevented by closing the avenue of infection, the eustachian tube.

The following conclusions may be based upon these figures:

1. That closure of the eustachian tube is harmless because it does not cause the loss of any function which has not already been permanently destroyed.
2. That it is a valuable therapeutic agent because by it alone half of the cases are cured and the rest so improved that it enables the radical operation to cure the other half.
3. That it is imperative, not only because the tube has assumed a perverted or pathologic function, but because the only other resource which is open to the patient is a radical operation, in which case the tube must be closed anyway.
4. That every patient should be given the benefit of this procedure, not only because the chances of cure are sufficiently high, but also because if cured thereby the hearing will not only be preserved, but improved.
5. That the figures here presented also justify the statement that on account of the difference in the effect upon the hearing, the radical operation should be regarded as unjustifiable in any case where there is useful hearing left, until a thorough trial has been given to the closure of the tube alone through the intact auditory passages.

DISCUSSION.

DR. FRANCIS P. EMERSON, Boston, congratulated Dr. Yankauer on having devised an effective instrument for clearing out the eustachian tube. He was in accord with Dr. Yankauer's views concerning the pathology of the eustachian tube, and with reference to the importance of stopping the reinfection of the tube. His experience, however, and the statistics which he had gathered from other operators at the Massachusetts Eye and Ear Infirmary, did not conform to the essayist's ideas concerning the permanency of the closure of the tube. In the condition to be dealt with there was a funnel-shaped cavity which was already infected, in which the lining membrane might be absent, and in which necrosis of the bony element might be present. The endothelial elements were of lowered vitality. Healing must take place either through granulation or through cicatrization. If there was healing through granulation there would first be inflammation and reaction. During this stage the tube was closed down. But if statistics were to be of value as to the permanency of the healing of such a tube, they must be taken two or three years after the operation. In order to try out this method he and Dr. Mosher had turned over all their cases to one of the assistant surgeons, Dr. Lougie, and his results were embodied in a paper read before this society in Atlantic City last year. Out of twenty-five cases examined it was found that the tubes were open, with discharge and granulations, in eleven cases; tubes closed, with ear discharging, in one case; tubes open, with ears dry, in eight cases; tubes closed, with ear dry, in one case. There were four cases in which cholesteatomata were present. These should not be included. During the last month, in order to follow the matter a step further, the speaker went over the records of the Massachusetts Eye and Ear Infirmary, and of the cases which had been operated upon by different operators and in which no skin grafting had been done, only twelve reported for examination. Of these twelve cases only one tube was closed. This was a case in which Dr. Mosher had introduced into the lumen a piece of cartilage. Over this piece of cartilage there was a thin layer of epithelium.

Dr. John F. Fairbairn, Buffalo, had tried the method in four selected cases, shortly after the publication of Dr. Yankauer's paper, and after Dr. Yankauer had demonstrated the method to him. He said selected cases advisedly, because he did not believe the procedure would be effective in the presence of a pathologic condition in the attic, antrum, or of the ossicles, but absolutely indicated in tubal suppuration. In three cases he succeeded in getting dry ears; in the fourth he got no result. The only difficulty encountered was the intense reaction which followed the operation. This reaction extended up over the promontory. He had since used Urbantschitsch's modification of the Yankauer instrument and method. This instrument was a rasp-like celluloid bougie. A small sized catheter was first passed through the extended canal to the aural opening of the tube, and through this the bougie was passed and the walls of the tube denuded. He had used this method successfully in five cases.

Dr. Yankauer, in closing the discussion, said he had been unsuccessful in obtaining a reply from Dr. Lougie to the circular letter which he had sent out. He was therefore glad that Dr. Emerson had referred to them. It is not correct to judge the operation by comparing cases in which the operation had been successfully performed with those in which it had been performed unsuccessfully. It had been shown by this investigation that in cases in which the tube was not closed after curettement, a larger percentage were improved in regard to the suppuration. It was likely that if none of these cases of Lougie's had been curetted, not so many would have been cured. If all of them had been successfully curetted and the tube closed, many more would have been cured.

Referring to Dr. Beck's remarks, he stated that it was necessary to remove the mucous membrane from the isthmus; because at this point there was an osteochondral synarthrosis; and at such a place, where cartilage meets bone, any irritation will cause thickening of bone, and hence here a bony atresia might be produced. That temporary closure of the tube could be produced by swelling of the mucous membrane seemed quite possible, and in such cases the tube would reopen.

The instrument of Urbantschitsch, to which Dr. Fairbairn referred, was a roughened celluloid bougie, and was introduced into the tube through the eustachian catheter, and was not intended to destroy the mucous membrane, but merely to set up an irritative inflammation.

The End Results of the Radical Mastoid Operation.

Dr. S. MacCuen Smith gave a short review of the evolution of the mastoid operation from Riolanus in 1649 to Stacke in 1891. In his practice he had considered the indications for the mastoid operation valid only in those cases that had utterly failed to respond to persistent nonoperative treatment, but he believes that the end results are probably much better, especially in point of hearing, when the operation is performed reasonably early.

In a large percentage of these chronic cases the middle fossa is found to be unusually low, the sinus in some instances being so far forward as to occupy the greater part of the antrum; hence, the advantage of an X-ray photograph to determine these points, more particularly the position of the sinus.

Dr. Smith sent letters to three hundred and thirty-four patients, requiring them to come for an examination as to the end results of the radical mastoid operation. Two-thirds responded. Suppuration had completely subsided in a large percentage of cases, and in those in which it still continued, its source was the tympanic cavity, being usually due to the eustachian tube becoming patulous. An examination of some of the cases which showed no discharge showed complete dermatization, an improvement in the hearing, entire absence of all head symptoms, and not the slightest accumulation of desquamated epithelium. The time of examination varied from two to fourteen years after operation, and the duration of the otorrhea before operation ranged from about one year to forty-seven years. The time for after-treatment for out-patients in the hospital service averaged three months. This period was considerably reduced for the private patients. The author had never seen an intracranial complication develop after a radical operation. The patients, almost without exception, improved in general health. Dr. Smith found frequent intracranial or labyrinthine involvement at the time of operation, unsuspected before, which almost invariably complicated the recurrent type of chronic discharge.

The cases ranged from one year to over fifty years in age, the largest percentage being between twenty and thirty. If the functional activity of the internal ear remained the same as before operation, the hearing of the average case should be as good after this surgical procedure as before. In thirty-two per cent of the operated cases the hearing is definitely better. In forty-nine per cent it remained relatively the same; of the remaining nineteen per cent, in eleven per cent the hearing decreased after operation, according to actual records, while in eight per cent it decreased according to the statement of the patient. The author, therefore, believed that the operation is not likely to result in an impairment of hearing.

Bell's palsy developed in three cases of the author's series before operation, and eight subsequent thereto. Two improved slightly and four entirely recovered, and two approximately so.

The radical operation should be regarded as a major procedure. Nevertheless, it is usually productive of a maximum amount of good and is therefore wholly justified. If chronic otorrhea has its origin in the mastoid antrum or lower cells, it can be relieved, as a rule, only by mastoid operation.

DISCUSSION.

DR. ARTHUR B. DUEL, New York City, emphasized the fact that in chronic otorrhea requiring radical operation two distinct types of cases, offering two distinct problems, were encountered. In the first class of cases the life of the patient was in danger because of the surgical condition present; in the second type there was an annoying discharge which repeated efforts had failed to cure. The end results in these two types of cases were naturally different. The statistics which had been formulated as to the length of time of healing had been confused with regard to these two types. Those who claimed that their radical cases healed in from five to six weeks, had operated in a class of cases in which the surgical condition consisted in a mass of granulations, without necrosis in the bone, and an annoying discharge. The cases which necessitated radical operation for the other condition required, in his experience, a longer time for healing—usually from eight to ten weeks, and, in many cases, six months to a year. He would never consider a case in which cholesteatoma was present as healed until at least two years had elapsed without recurrence. The patient, in an uncomplicated case, could be considered out of danger as soon as the operation had been performed. In the cholesteatoma cases the patient was in some danger, though infinitely less after operation, until recovery was complete. For a long time he endeavored to skin graft all his cases, but in his experience had found it better to do the skin grafting in the third week after the operation—that is, after the cavity had been covered over with granulations. He thought it much safer surgery to leave out the primary skin graft in all cases. In cases in which exposed dura over the middle fossa or sinus was perfectly healthy, it might be considered safe to cover immediately with a skin graft; but who could be certain in a suppurating wound, that he was not bottling up virulent organisms? In many cases it was necessary to expose large portions of dura which were covered with a mass of suppurating granulations. One could hardly doubt in such cases that there was great danger in covering this immediately with skin graft, lest the organisms present set up an active inflammation either in the exposed dura over the middle fossa or sinus. Even allowing that antiseptics strong enough to sterilize these areas were used, they would at the same time render the granulations unfit for receiving skin grafts.

Dr. E. B. Dench, New York City, referring to the effect of chronic purulent otitis media upon the general health, cited two cases in which there had been marked improvement in the general health following the radical mastoid operation. One patient had gained nine pounds in two weeks. This chronic aural suppuration produced a low grade toxemia, and it was often difficult to determine just what was the matter with the patient thus affected. The patient who gained nine pounds was supposed by the family physician to have malaria. As to the effects of the radical operation on the hearing, he was glad to see that Dr. Smith's statistics tallied with those which the speaker had presented at the Otological Congress at Bordeaux. If hearing was very poor, and there was no labyrinthine complications, hearing would be improved after radical operation. If hearing was only moderately impaired, with no labyrinthine involvement, it would remain the same. If hearing was very good, the patient should be told that the operation would probably make the hearing worse. He had not been able to decide upon the two types of cases mentioned by Dr. Duel. He thought every case with chronic middle ear suppuration in danger. It could not be said that one case was in danger and another not. Until one had cleared out the middle ear and tympanic cavity and mastoid, and could see the sinus and middle cranial fossa, one could not say there was no danger. The primary skin graft had been successful in his hands. He had never seen a case in which the skin graft caused trouble unless the sinus or the dura were very badly diseased. He had grafted over these areas repeatedly without trouble. Secondary grafting had not been so successful in his hands. He saw no reason for subjecting a patient to two anesthesias. Two weeks was the shortest time in which he had had dermatization. This was almost the invariable rule in both private and hospital cases, the tympanic cavity, roof of the tympanum, and entire mastoid cavity being absolutely dermatized and dry in ten days or two weeks. He had seen many patients two years after operation and had found the cavity perfectly dry.

DR. GEORGE F. KEIPER, Lafayette, referred to the fact that life insurance companies refuse to accept persons with chronic otitis media, and for that reason he always advised such patients to submit to radical mastoid operation. He thought this advisable, not only for the preservation of life, but in order to enable the individual to occupy a more satisfactory position in life.

DR. JOHN W. MURPHY, Cincinnati, knew of no class of cases that placed more responsibility upon the doctor than cases of chronic otitis media, not only with regard to life but also as to hearing. He recalled a case in point, that of a young woman, thirty years of age, who had a deaf mother and sister, and who, in childhood, had had double otitis media. The left ear had ceased to discharge, but had no membrane except scar tissue. She had applied for treatment for a rather offensive discharge from the right ear. She had no hearing in the left ear and a chronic condition in the right, with gradually decreasing hearing. He could not promise that the end results would be better after operation. She consented to the radical mastoid operation. At the end of two weeks the discharge in that ear stopped, but at the end of two days she developed left sided paralysis. If that had developed on the right side, he would have thought the operation had something to do with it. It was a strange coincidence that a patient should have Bell's palsy on the side of the ear not operated upon, two weeks after the operation.

DR. JAMES E. LOGAN, Kansas City, spoke of cases in which, after thorough operation, with the cleaning out of all necrotic tissue, there were still recurrent attacks. In many such cases he had been able to give permanent relief by cleaning out the vault of the pharynx, which may have been the seat of infection, from time to time. He believed in many cases of chronic suppurative otitis the vault of the pharynx was the cause of the trouble.

Dr. Frank Allport, Chicago, had been in the habit of estimating his cases, with reference to hearing, in accordance with Dr. Dench's formula. In the large majority of cases the radical mastoid operation gave good results so far as hearing was concerned. For the last five years he had used gauze only for the first few days, substituting the gauze packing in a week's time with boracic acid powder, filling the cavity almost completely with the boracic acid powder and covering this over with a little gauze. Healing took place more quickly under this form of packing than with anything else. He agreed with Dr. Logan with regard to the part played by the pharynx in the clearing up of these cases. The end results would be better if all paid better attention to the pharynx, particularly to the clearing out of the fossa of Rosenmüller. He did not employ skin grafting in all his cases. Whether one resorted to grafting or not, Dr. Dench's results with this method were notable. He had formerly employed the Ballance method, and also Dr. Hammond's method, but latterly, when he used a graft at all, he followed Dr. Dench's plan.

Dr. George F. Cott, Buffalo, said one sometimes had the opportunity of making postmortem examinations of cases in which the radical mastoid operation had been performed. He had had one hundred and eighty radical mastoid operations. One patient had had a discharge for forty years, and she was forty-four years old. He did a radical operation, when he found a hole in the dura one-eighth of an inch in diameter, through which the probe passed one and one-quarter inches. He dropped the probe down easily, slit this open, and did nothing more. The patient did well. At the end of two years she got sick suddenly, and died in two days. Postmortem examination revealed basilar meningitis, with meningococcus infection. In another case there was acute middle ear suppuration, with intense pain for a week. He operated; the patient did well for two days, then the temperature, apparently normal, went up to 104.5 degrees, then down to somewhat below normal. The patient died. The subarachnoid spaces were found, upon postmortem examination, to be entirely filled with blood, in different stages of clotting. In the third case a girl had acute otitis media of six weeks' duration. At operation the entire mastoid bone was found to be sclerosed. She had had pain in her eyes since she was very young. She apparently recovered from the operation. He saw her at intervals for six months. She died suddenly, having considerable pain at the base of the brain. Postmortem examination revealed a cerebellar abscess as large as the little finger. She had not had a symptom other than the pain. Two abscesses were found on the parietal lobe of each side.

Dr. Thomas J. Harris, New York City, said it had seemed to the program committee that Dr. Smith's paper was exceedingly timely. Judging from the discussion, it would seem that the paper had given the impression that there was nothing but the favorable to be said concerning the mastoid operation. It was his own observation, in this country and abroad, that this was not the case, that such were not the results that were being obtained. It was for this reason that Dr. Smith had been invited to discuss this subject. One had only to go into a clinic in any large city to see patients treated week after week for chronic suppuration, to see case after case of facial paralysis going in for dressings. Was it fair to consider this operation from the results of brilliant operators, or should it not be considered from the point of view of the majority of operators? As Dr. Cott had said, there were cases of death. Was death, in these cases, due to the mastoid, or was it coincidental? The mastoid operation was not performed entirely for chronic suppuration; it was performed frequently as the next step in an intracranial operation. He wished that there might at some time be a referat, as had been done in Dr. Yankauer's paper, so that the results of those having a small number of cases might be obtained. There was a feeling that the radical mastoid operation was being overdone, that it was done too quickly. Was that the case? After reviewing the statistics, could it be said that the results for the last fifteen years have been good?

Dr. Smith, in closing the discussion, said when skin grafting was first introduced he had used it with not very satisfactory results. He discontinued the method for a number of years, and then, stimulated by Dr. Dench's results, he had taken it up again, but could make no report on the cases so treated because they were too recent. He employed Dr. Dench's method, using one large graft and two smaller ones, all primary grafts. He had had few cases of Bell's palsy, three of which occurred previous to operation. In two cases he was called out of town on the day appointed for the radical mastoid operation, and that night Bell's palsy developed. If he had operated at the appointed time the palsy naturally would have been attributed to the surgical procedure. Deaths following the radical mastoid operation were, in his opinion, due to complications. He had never seen a death occur in an uncomplicated case.

Vertigo of Labyrinthine Origin Following a Chronic Suppurative Otitis Media With Cholesteatoma. First—Simple Mastoid With Curettage of the Middle Ear and Eustachian Tube. Second—Radical Labyrinth Exenteration.

DR. FRANCIS P. EMERSON: The otologist is particularly interested in those labyrinth cases which, in their final analysis, confirmed by operation, may be readily labeled pathologically, and yet in their clinical manifestations may have presented a reasonable doubt as to the surgeon's attitude in regard to immediate operative interference. The relation of the vestibular apparatus to the brain demands exact diagnosis and immediate surgical intervention in all pyogenic invasions of the labyrinth, if life is to be saved. The case outlined is offered as one with delayed symptoms, and one which was perhaps less clear clinically than many.

The patient, male, aged sixty-two years, was admitted to the Massachusetts Charitable Eye and Ear Infirmary, August 11, 1913, with a diagnosis of otitis media suppurativa chronica of the right ear. The case is summarized as follows: The patient is a man prematurely old. There is a perforation of the septum. His wife has had two miscarriages. The hearing test for his good ear—that is, the left—the writer has found to be strongly suggestive in other cases of lues—i. e., a low note, but little, if any, raised, a positive Rinné, with marked lowering of bone conduction, and the whispered voice reduced to 1/25 or 1/35. In this case there was a negative Wassermann, but no examination was made of the cerebrospinal fluid. His eyes and a physical examination did not give evidence of specific disease. Could lues then be eliminated as a direct or contributory factor in this patient's symptoms?

The right ear gave a history of discharge for thirty years, and in addition there was a cholesteatoma, which, as is well known, is quite apt to invade the labyrinth. There were granulations in the posterior quadrant. The hearing test done several times always showed vestibular irritation when the vertical canals were in the plane of rotation. Unfortunately the difference in the after-nystagmus between the turning to the right or to the left was not recorded. The fistula and caloric tests were negative, and there was no nystagmus. The hearing test, done by two different observers, showed total deafness for the right ear, except a bone conduction of three inches. Can we say that this then was a case of diffuse purulent latent labyrinthitis, and that the apparent vestibular reaction was compensatory? According to Ruttin, this does not occur unless a latent purulent labyrinthitis has existed for a long time, and has been followed by a complete ossification of the labyrinth or sequestration. In this case there was no clinical history of vertigo until five weeks before admission, and our operative findings showed the horizontal canal free, while the superior and posterior contained a grayish red, granular, nonadherent detritus. There was no organized tissue or evidence of ossification present. The second day after operation we have a clear, serous discharge resembling cerebrospinal fluid. There was no facial paralysis, and no sequestrum was found. On the other hand, the turning test was always positive.

Have we any evidence to suggest that this was a case of diffuse serous secondary labyrinthitis? The onset of the vertigo favors this supposition, and is confirmed by the hearing test, which shows a remnant of vestibular irritation present. The operative findings are also consistent with this diagnosis; on the other hand, the caloric and fistula tests were negative, and there was no nystagmus at the time the patient was examined. The caloric reaction we know may be lost. If we assume that the portal of entry was originally by way of the oval window, could we not explain the failure at this time to obtain a positive fistular test by the presence of granulations in the posterior quadrant of the middle ear? We recognize that such an invasion is more often followed by a purulent labyrinthitis; but three cases of fifty reported by Ruttin were followed by the serous form. There remains then to be accounted for the absence of nystagmus. This is a symptom of irritation, and if of the second degree, it might readily have been wanting at the time of observation. In the light of our symptomatology and the operative findings, the writer is inclined to consider the case one of a diffuse serous secondary labyrinthitis, as affording the only consistent diagnosis. For those who accept this interpretation, the division into two stages of the operative procedure is perfectly consistent. If this diagnosis is accepted, we must assume that there were no well defined symptoms immediately following the mastoid operation, but that if present at all, they were but little marked, and only developed as a pronounced diffuse serous secondary labyrinthitis on the sixth or seventh day.

DISCUSSION.

Dr. E. B. Dench, New York City, thought it a mistake to follow the foreign school in making a classification of these cases into serous, suppurative, or perilabyrinthitis. Neumann, Ruttin, and others had said if a diagnosis was made of one or another kind, one must do one or another kind of operation. He thought Dr. Emerson's plan of treatment in the case under consideration the proper one. He had had cases in which, if he had followed the foreign school, he would have killed his patient. The results of the German operators did not show that their operative recommendations were always warranted.

Dr. Norval H. Pierce, Chicago, corroborated what Dr. Dench had said with referenec to the foreign school and their methods. He cited a case, which had been under his observation at the Illinois Eye and Ear Infirmary, of acute otitis media, with obvious mastoid and labyrinthine involvement. The woman had had repeated attacks of discharge from the ear, extending over a number of years, each time running an apparently normal course, until the last, when swelling and pain appeared over the mastöid. She complained of great dizziness. The caloric reaction was negative, as to both heat and cold. There was nystagmus to the opposite side. She was absolutely deaf in the diseased ear, as proved by Neumann's apparatus. There was no fistula. Here was a case for immediately going into the labyrinth, according to the German school. He had so completely abandoned their ideas that he had probably swung to the opposite extreme. He did not open this woman's labyrinth, and she recovered. This was one of six or more similar cases which had come under his observation during the past year. He did a simple mastoid operation. It was impossible to differentiate between suppurative destructive labyrinthitis and serous labyrinthitis which was capable of recovery. The cases which he had observed for some length of time had recovered function so far as hearing was concerned. He believed one should never do a labyrinth operation when symptoms of meningitis were present; to operate under such circumstances would have very little effect.

Dr. Emerson, in closing the discussion, said his object in presenting this case was to bring out just such remarks as those of Dr. Dench and Dr. Pierce. These cases should have a preliminary mastoid operation, and the labyrinthine operation only in the presence of evident signs.

The Labyrinth Operation—The Frequency With Which It Is Demanded as Determined by Hospital and Private Statistics Extending Over a Period of Ten Years.

Dr. Edward Bradford Dench stated that it was the object of his paper to show by statistics the relative infrequency with which labyrinth suppuration occurs as a sequel either of acute or chronic middle ear involvement. Between 1904 and 1915 less than one-tenth of one per cent of the cases of middle ear involvement at the New York Eye and Ear Infirmary were subjected to the labyrinth operation.

Dr. Dench's own records, from May, 1905, to May, 1915, show six hundred and fifty-nine cases of acute mastoiditis and secondary mastoiditis operated upon, five hundred and thirty-three cases of chronic middle ear suppuration subjected to the radical operation, twenty-three cases of brain abscess, thirty-three cases of meningitis, and thirty-seven cases of sinus thrombosis. From 1907 to January 25, 1915, the same records show twenty-two cases in which the labyrinth operation was performed. These records would show that a labyrinthitis, demanding operation, is a most infrequent complication of a middle ear lesion. Labyrinthitis occurred in only two per cent of the operated cases of middle ear suppuration.

Dr. Dench called a removal of a portion of the labyrinth a "partial" operation, and the free opening of the horizontal semicircular canal, the vestibule and the cochlea, thus securing complete drainage of the entire labyrinth, the "complete" operation. He performed the partial operation in ten cases and the complete operation in twelve cases. Of the cases in which the partial operation was performed, seven were cured and three died; of the complete operation, seven were cured and five died. Of the three fatal cases of partial operation, one died of malignant disease and two of meningitis. Of the five fatal complete operation cases, four died of meningitis and one of a pulmonary thrombus secondary to a jugular thrombosis. In two of the remaining fatal cases the operation was performed for the relief of an existing meningitis.

Dr. Dench concluded that the necessity of the complete labyrinthine operation is comparatively infrequent, and that the operation should only be undertaken in the presence of very definite symptoms, pointing to either acute labyrinthine involvement or an acute exacerbation of a previous labyrinthine suppuration. When these symptoms occur, operative interference is imperative, and must be prompt if it is to be successful. The expectant plan of treatment will probably be followed by the best results in cases of circumscribed labyrinthitis, or of a purulent labyrinthitis which is quiescent.

DISCUSSION.

Dr. George W. Mackenzie, Philadelphia, disagreed with Dr. Dench regarding the treatment of cases of circumscribed labyrinthitis in the semicircular canal by curetting and nothing more. Of course it was desirable to preserve the hearing as much as possible, but by curetting in the neighborhood of a fistula one would be apt to spread the labyrinthitis. He had done this is one case, with loss of function in the horizontal position, but with retention of function in the vertical canals. In cases of labyrinthine suppuration, with positive evidence that the labyrinth was dead, the labyrinth operation should be done at the same time that the mastoid operation was performed, otherwise the case was apt to terminate fatally. He had reported fifty-five cases, not his own, in which there were two fatalities. One was a case of Neumann's, and one Alexander's. Both these fatalities were due to the fact that the radical mastoid was done and the labyrinth left untouched, when the labyrinth should have been operated radically. There was some doubt in the minds of these surgeons as to the diagnosis of labyrinth suppuration before the operation on the mastoid. These two errors were committed early in the experience of these men, at a time when the knowledge of the labyrinth was more limited than it is now. His present conviction concerning the subject of labyrinth suppuration is that we should not attempt any operation on the mastoid without at the same time draining thoroughly the labyrinth. Furthermore, that no operation on the mastoid or labyrinth should be attempted until a most careful examination of the labyrinth by all known tests had been made and a positive opinion arrived at concerning the condition of the labyrinth. Circumscribed irritative conditions of the labyrinth (congestion) without the fistula sign did well with mastoid operation alone. Circumscribed destructive conditions would always tax the skill of the surgeon as to what was best to be done. Each case would need separate consideration, as there was no fixed law by which one should be governed in all cases.

DR. ARTHUR B. DUEL, New York City, thought it interesting to note the change of attitude with reference to these cases of suppurative labyrinthitis. When the Vienna school first promulgated their ideas, their plan was pretty generally adopted. No doubt many mistakes were made as a consequence. The general opinion among surgeons of the present time was to regard acute cases, whether suppurative or not, as unfavorable for operative intervention. These cases should never be operated in the acute stage unless there were evidences of a concomitant meningitis. It was much better to wait; for, if operated upon immediately, they were much more apt to die, by precipitation of the very complication they were trying to avoid, rather than to recover. He had shared with Dr. Pierce the rather hopeless view of meningitis until last year, when he succeeded in curing a case by means of the Neumann operation and drainage at the internal auditory meatus, accompanied by repeated spinal punctures, drawing off twenty to thirty cubic centimeters of milky cerebrospinal fluid, at the same time injecting back about forty grains of urotropin in fifteen or twenty cubic centimeters normal salt solution. At each subsequent tapping of the cord, done at intervals of one or two days, six times, the fluid became more and more clear, until the patient finally recovered. He shared the belief of the majority, that in chronic cases if at the time of operation the gross appearances indicated the presence of a fistulous opening and necrosis, it might be necessary to open the labyrinth; otherwise it was better to wait for further symptoms.

DR. MACKENZIE added that many cases of supposed labyrinthine suppuration of ten years' standing might present all the symptoms of labyrinthine suppuration, and yet there might never have been this condition, but, instead, a chronic labyrinthitis of the plastic type. Unless, therefore, Dr. Beck, or someone else, had seen the case at the very onset, with complete loss of function, it could not be pronounced a case of labyrinthine suppuration. There must be typical signs of suppurative labyrinthitis. There was, with suppurative labyrinthitis, sudden, complete loss of hearing, loss of equilibrium, with negative findings by caloric tests, by galvanism, and by turning. Answering a question by Dr. Pierce concerning the method of differentiating between serous and suppurative labyrinthitis, the speaker said it was indeed difficult to make the differentiation early. Headache and fundus findings were guides. When present they suggested a suppurative type of labyrinthitis.

Dr. George L. Richards, Fall River, asked whether the discussion concerned the perilymph or endolymph channels, whether within or without the membranous labyrinth. It made a great difference.

Dr. Norval H. Pierce, Chicago, said the functional symptoms of serous and suppurative labyrinthitis were the same, and one condition could not be differentiated from the other. The only light thrown on the subject was that given by the meningeal complications. It was better not to operate until the meninges were involved.

Dr. Dench, in closing the discussion, said, in answer to Dr. Richards' question, the subject involved both the perilymph and the endolymph, and was within the bony capsule. It could not involve the endolymph without involving the perilymph. That was just the hair-splitting question the German school had raised. He could not agree with Dr. Mackenzie with reference to the treatment of circumscribed labyrinthitis. He believed it wise, in the presence of a fistulous tract, to go in with the curette, where this could be done without breaking down nature's barriers to the course of infection. He removed all necrosis with a small curette.

Report of an Unsuccessful Labyrinthectomy for Relief of Distressing Tinnitus Aurium.

Dr. Arthur B. Duel: Tinnitus aurium of such distressing character as to produce insanity, or to drive the sufferer to suicide, is, fortunately, a rare occurrence. The occasional case, in which every effort has failed, and where sedatives have become useless, except for the immediate time being, present as sad a spectacle as the most hopeless alien. The few cases that have been reported in which major operations, like division of the auditory nerve or destruction of the labyrinth, have been tried for their relief, have met with so little success that no one would be tempted to undertake another, except under great stress of circumstance, or some peculiarly hopeful prospect, as in the case recorded.

The patient, a woman, sixty years of age, married, had slight deafness following typhoid fever at the age of eighteen years. At the age of thirty-five she first noticed noises in the head, described as escaping steam. Hearing became gradually impaired, and the noise persisted. She was variously treated by different specialists, without relief. She finally came under the observation of Dr. Fielding O. Lewis, of Philadelphia, in 1912. At that time she could hear slightly a watch in close contact with the left ear, and a whisper at about six inches. Local measures failed to improve the condition. She now complained of a monotonous humming around the outside of her head, with intervals of a thumping sensation on the right side of the head. The division of adhesions about the ossicles of the right ear, lumbar puncture, ossiculectomy of right ear, all gave but temporary relief, the tinnitus becoming more and more distressing, necessitating the administration of small doses of morphin. Destruction of hearing on the right side was suggested, but consultants advised against this procedure. Dr. Duel was then called in consultation. After noting all the necessary tests, and after discussing the probable outcome, it was decided to ablate the right labyrinth. A complete labyrinthectomy utterly failed to influence the tinnitus. The special feature of the case which caused its being placed on record was the fact that three months after the labyrinthectomy Dr. Lewis found that the patient still had hearing on that side. The tinnitus was no better. The most carefully conducted tests made by Dr. Duel confirmed this unusual finding.

Six months after the first operation a second was performed. Just before operation it was again demonstrated that the patient could repeat words shouted in the operated side, the unoperated side being stopped by a noise apparatus. At this operation the bone wax which had been employed at the first operation to stop the flow of cerebrospinal fluid was removed from the cochlear cavity, which was apparently clear of any vestige of the cochlear apparatus. Thorough curettage of the cochlear cavity was performed, and the vestibule, which was entered from behind, and the ampullæ of the semicircular canals, were destroyed. No bone wax was used on this occasion. The wound healed and was dry for several months at the time of the report.

The important phenomenon observed in this case might cause one to question the theory of Helmholtz with reference to functions of the labyrinth. Does it support that of Shambaugh? At any rate, it should stimulate further investigations of the physiology of sound production.

DISCUSSION.

Dr. George E. Shambaugh, Chicago, said that the first question of importance in this case was to determine the probable cause of the ear trouble. From the history of the case alone, deafness coming on insidiously in middle life, associated with annoying tinnitus, it would appear most likely to be one of otosclerosis. This at once offered a probable explanation for the most interesting feature, namely, that the patient appeared to hear in an ear where the cochlea had been completely destroyed. The explanation that suggsted itself was this. In otosclerosis the symptom of paracusis Willisii was usually very marked. In these cases it was not unusual for a patient who had great difficulty in hearing the voice to find that in the presence of a noise—as, for example, on a railroad train—he could hear even better than a normal hearing individual. Now in the case here reported, where the cochlea in one ear had been destroyed and a noise apparatus was applied to the opposite ear, it seemed not unlikely that through the phenomena of this paracusis Willisii the patient might be able to hear even where no hearing could be detected when the same test was applied to a normal ear.

The generally accepted view was that sound perception takes place exclusively in the cochlea. If this be true, then an injury even less extensive than in this case, it would seem, must result in a total destruction of hearing in that ear. If it be true that the patient did actually hear in the operated ear, he believed it must be accepted that the perception took place in the vestibular apparatus. He did not believe that any theory of sound perception which placed this function in the cochlea would account for hearing in an ear which had been subjected to the operation performed by Dr. Duel.

The annoyance from tinnitus aurium was much greater in some cases than in others. Some of this difference was to be accounted for by the difference in the general nervous make-up of individuals. A subjective noise which would cause only moderate annoyance to one person would in another more nervous individual almost drive to suicide.

Dr. George W. Mackenzie, Philadelphia, thought it unfortunate that careful tests were not made before anything was done, as it might then have been determined whether the lesion was in the conducting or the perceptive apparatus. It was possible in nerve lesions to have tinnitus aurium. Two cases had been reported eight years ago, in the *Wiener klin. Woch.*, by Dr. Alice V. Mackenzie, which were of syphilitic origin. He suggested that in Dr. Duel's case the condition was a post-syphilitic lesion. He did not believe the patient heard. He thought the combination of the speaking tube method with the noise apparatus would have given a different result.

Dr. Fielding O. Lewis, Philadelphia, said the patient still insisted, with the use of the noise apparatus, that she could hear. Dr. Duel had made a handsome wager that she could not hear after the first operation, but by the careful tests described had demonstrated himself a loser, both to himself and all others present. After the ear became dry, following the second operation, the speaker had tested it again with the noise apparatus, and she still said she could hear sounds but could not determine words. He had her daughter, whom she could hear best, stand by her, without speaking, and the patient again insisted that she could hear sounds but not words. She had been taken to a sanitarium. She had never had paracusis Willisii. Wassermann test was negative, and she had been given iodids. Other tests were negative.

Dr. Duel, in closing the discussion, said the functional tests had been as elaborate as they could be made. If, as might be assumed, a portion of the cochlea had been left at the first operation, the wax, impinging upon that, might have carried the sound waves in, if Dr. Shambaugh's theory is correct. If a remnant of the cochlea had been left at the first operation, it had probably been removed at the second. The curettage of the vestibule in the first operation had not been done as thoroughly as that of the cochlea. In the second the whole labyrinth had been removed. The point he raised was whether destruction of the cochlea destroyed hearing.

Experimental Studies of the Effect of Various Atmospheric Conditions on the Upper Respiratory Tract.

DR. GERHARD H. COCKS: The purpose of the experiments upon which this contribution is based was to add to the knowledge concerning the exact place of atmospheric changes of temperature in the production of respiratory disease. In order to accomplish this, direct observations were made upon the respiratory tract under conditions which could be absolutely controlled as to temperature, humidity and air movement.

The observations were conducted under the auspices of the New York State Commission on Ventilation, who provided for the purpose two adjoining rooms, approximately ten by fourteen feet each. These rooms were so arranged that any degrees of temperature and humidity could be secured and recorded. Room A, the Control Room, was fitted with white tiled walls, and with machinery for controlling atmospheric conditions in both rooms, viz., fans, air washer, heating stacks, steam lines and ducts. Room B, the Observation Room, was also fitted with white tiled walls, covered with cork board, cemented and impervious. The two rooms, were adjacent, with a window in the common wall. Metallic mirror records were made of the results of the clinical examinations of the nasal passages in practically all experiments except those made in the hot moist room (86° F., eighty per cent relative humidity).

The experiments were made on the following divisions:

1. Series A—Examination of one hundred and thirty-one cases, changing from one atmospheric condition to another.
2. Series F, Studies in Air Movement.—Examination of seventy-seven cases, changing from one atmospheric condition to another, with fans blowing directly on the face.
3. Series B, Skin Reflexes.—Examination of fifty-seven cases, with body in one room and head in another room having a different temperature.

4. Series C, Skin Reflexes.—Examination of forty-two cases, with the body exposed to one temperature and the feet immersed in a water bath of a markedly different temperature.

5. Series G, Studies in Workers Exposed to Dry Heat.—Examination of forty-five cases of workers in dry heat (firemen, stationary engineers, boiler workers, etc.), on going from one temperature to another.

6. Series G, Studies of Outdoor Workers.—Examination of fifty-eight cases of outdoor workers (truckmen, drivers, etc.) on going from one temperature to another.

7. Series G, Studies of Cases of Atrophic Rhinitis Corresponding to Workers in Moist Heat.—Thirty-nine observations on cases of atrophic rhinitis on going from a hot dry room to a cold room, and vice versa. It was found that a large proportion of workers in hot, moist rooms (steam laundries) suffer from atrophic rhinitis. Owing to the difficulty of securing employees of laundries who could be induced to give the time required for the experiments, other cases of atrophic rhinitis were substituted for laundry workers.

8. Series G.—Examination of forty-six laundry workers (while on duty in two steam laundries in New York City).

9. Experimental observations on the larynx and trachea of dogs subjected to peripheral stimulation by means of heat and cold.

Summary and Conclusions.—Clinical experiments demonstrated that distinct changes in the mucous membranes of the nose result from changes of air temperature and humidity. In the majority of instances the reaction is one of increased swelling, moisture and redness from heat, and the reverse from cold. The effect of air blown directly upon the face by fans greatly modifies the changes observed. On going from the cold to the hot room with fans there is a decrease in the size of the inferior turbinate and in the amount of moisture. The characteristic change on passing from the hot to the cold condition with fans is an increase in the turbinates and secretion. It was further observed that moist heat produces greater changes than dry heat, while the highest percentage of cases of atrophic rhinitis was found among long-time workers in hot, moist rooms (steam laundries).

The window and foot bath experiments tend to show that the reaction of the nasal structures to atmospheric changes is primarily direct and local, and not reflex, although evidence on this point is inconclusive. It must be remembered that turbinate reactions are very delicate, and that the changes observed were by no means constant. It would appear, however, that the reactions in the nasal mucous membranes produced by changes in atmospheric environment are too frequent and too definite to be disregarded. Consequently, he was convinced that the theory that bacterial infection is the sole cause of catarrhal inflammations of the upper air passages is not tenable, since the changes produced by environment must materially affect the incidence of infection.

DISCUSSION.

DR. WOLFF FREUDENTHAL, New York City, considered the effect of atmospheric conditions on the nose and throat a very important question, one in which he had been interested for more than twenty years, and upon which he had written a great deal. When he first came to this country he noticed that he could not breathe as well as in Germany, and that when he went into hot apartment houses he could hardly breathe at all. He began then to investigate the amount of humidity and the temperature in these houses. The normal amount of the relative humidity should be fifty per cent, though one could live comfortably in an atmosphere of forty per cent. But in winter it went below twenty per cent. He could not agree with the essayist as to the influence of moisture in the atmosphere. The experiments were highly interesting, but not conclusive. In New York and the New England States it was not the moisture of the surrounding waters the effects of which one felt. The majority of people spend daily twenty-three hours at least in very hot and very dry rooms and not at the sea shore. He believed that dry atmospheres were conducive to dry rhinitis, and that those who live in moist atmospheres do not have this. He was surprised at Dr. Cocks' statement with reference to laundry workers having dry rhinitis. The steam, as Dr. Cocks had said, came directly up from the manglers. If applied to the hands it would burn them, and the same, applied to the mucous membrane, would cause destruction of the same. The effect was not one of moisture, but of burning. It was surprising to see how well some people could breathe with swollen mucous membranes, whereas others would complain of being unable to do so with dry and wide nasal chambers. The latter class could not assimilate the air well. Experiments along this line had been made at the University of Kiel, and Freudenthal's views corroborated in every point. At New Bedford Station, forty miles outside of New York, experiments had been made with reference to tuberculosis, and it was surprising to see how much effect dryness of atmosphere had on these patients.

Dr. Arthur I. Weil, New Orleans, said this paper explained the statement, made in conversation, of one of the members of the society from Denver, to the effect that he practically never saw a case of atrophic rhinitis in that city. In New Orleans, on the contrary, the speaker saw more of this condition than of any other. Dr. Cocks' investigations showed why this should be the case. He could bear out Dr. Freudenthal's contention with reference to these patients with atrophic rhinitis complaining of lack of air. He had formerly thought that when patients complained of inability to breathe through the nose he would find hypertrophic rhinitis; now, however, he was equally prepared to find atrophic rhinitis.

Dr. George F. Cott, Buffalo, called attention to a forthcoming article in the Annals which would bear upon the subject under discussion. Dr. Cocks did not state whether the air used in the experiments was pure or contaminated. The air one ordinarily breathed was contaminated. In experiments with the air from schools in Brooklyn there had been collected five pails of dust in five days from the air the children breathed. This was a powerful factor in determining the results.

Dr. Thomas J. Gallagher, Denver, believed that among the rhinologists of Denver it was the consensus of opinion that cases of atrophic rhinitis developing there were seldom encountered. The vast majority of cases seen there were in people who had lived in a low, moist climate and in whom atrophic rhinitis had already developed before coming to Colorado. Subjects of atrophic rhinitis were more uncomfortable in Denver, owing to the diminution of the humidity of the air. The turbinate bodies must throw out more fluid, and their inability to do so resulted in an increase of crust formation. However, he had seen many cases of vasomotor rhinitis develop in the high and dry climate. The cause of this was not yet known, and the disease was peculiarly rebellious. It was certainly not due to the flora in different altitudes, as it occurred throughout the year without any amelioration, the patient remaining in the same place. He had seen remarkable results from sending the patients to the sea shore. The effect was very complex, but the good results probably come through the local influence of the air upon the nasal membrane plus the general effect upon the blood vessels and nervous system.

Dr. Robert Levy, Denver, did not believe atrophic rhinitis occurred rarely in Denver. It was mainly a disease originating in childhood. The cause was still in doubt. As a matter of fact, children with atrophic rhinitis were found in Denver, but, in his experience, the condition was not confined to children. It was more common in children than hypertrophic rhinitis. His clinical assistant, Dr. Cooper, was making a study of cases of atrophic rhinitis in children and adults, and he had enough cases to make a presentable showing. He had seen enough cases in the clinic alone, aside from those in private practice, to warrant his going on with the study. It was not, however, as frequently found in Denver as elsewhere.

Dr. Cocks, in closing the discussion, answered Dr. Cott's question in regard to dust by stating that in his opinion the results of the tests made in the experimental chambers were not influenced by contamination with a dusty atmosphere. The physical conditions in the experimental chambers were practically ideal. Nobody was allowed in the rooms except the observers and the subjects of the experiments.

Hemilaryngectomy for Malignant Diseases—Description of New Operation With Report of Six Cases, All Recovered.

Dr. John E. Mackenty presented a specimen by which he endeavors to make the closed operation as safe as the open one, without the difficulties and defects of the latter. Hemilaryngectomy stands midway between total laryngectomy and thyrotomy as a surgical choice. Thyrotomy takes the early cases of laryngeal cancer, hemilaryngectomy the later ones, and total resection the latest (or, as one might better say, the too late) ones. The statistics of recurrence are in corresponding ratio, thyrotomy leading and total laryngectomy bringing up a sorry rear. Gluck's method of hemilaryngectomy is a roundabout and tedious one, and has furthermore the disadvantages of permitting wound secretions readily to enter the trachea, and of giving some trouble later in converting the fissure into a potential tube. Yet a primary closure of the larynx without some way of safeguarding the trachea against wound secretion would be a grave surgical blunder. These considerations, therefore, prompted the author to attempt a modification in the accepted technic.

Operation.—The larynx should be first opened for inspection, if there is a question as to whether thyrotomy or hemilaryngectomy is indicated. Then, if hemilaryngectomy is elected, the trachea is opened just above the sternal notch. One incision is made for the laryngectomy and another for the tracheotomy. The first incision begins well up under the chin and extends to a point just below the cricoid ring in the center line. This is deepened until the thyrohyoid membrane, the thyroid and the cricoid cartilages are exposed. Care is taken not to separate the tissues laterally. The larynx is then opened in the center line, its interior cocainized and inspected. Blunt hooks or retractors are used in order to avoid injury to the edges of the cartilage, especially on the healthy side.

The trachea is next opened as low as possible. The second incision is here made over the end of the sternal notch and up to within one inch, or more, if possible, of the first incision, leaving a liberal bridge of tissue between the two incisions. The trachea is opened and a large tracheal cannula inserted. Into this is fitted a rubber tube, eighteen inches long, through which the anesthetic is continued. Before inserting the tracheal cannula, two stout silk threads are passed through the edges of the tracheal incision, one on each side, and laid one on each side of the neck. The wound is then closed fairly close up to the cannula. The object of the silk threads is to control the tracheal opening in case the tube should get displaced or come out. Traction on these threads opens the trachea. This precaution should always be taken in low tracheotomies.

The larynx is again opened with blunt hooks or narrow retractors. The trachea is cocainized and loosely packed with gauze. The patient is placed in a semi-Trendelenburg position. The skin, cervical fascia, and pretracheal muscles covering the diseased half of the larynx are carefully dissected away from the cartilage, taking care to keep them all in one flap and not to injure the inner surface, which is composed of the pretracheal muscles, as this surface is to form the new laryngeal wall. This flap is gently held away while the removal of half of the larynx proceeds. The thyrohyoid membrane is slit laterally and the trachea divided below the cricoid ring on the diseased side. Iodoform gauze is packed into the pharynx, and is removed through the mouth when the operation is near completion. The loosened half of the larynx is now retracted, well away from the center line. If the mucous membrane over the posterior commissure and arytenoid is healthy, it is saved and lifted away from the cartilage beneath. This mucous membrane dissection is carried backwards over the posterior surface of the arytenoid and down towards, and sometimes into, the anterior aspect of the mouth of the esophagus.

The diseased half of the larynx is now removed in the usual way, being careful to save the mucous membrane flap outlined above. All bleeding points are controlled by catching only the vessels and by tying with an 00 iodized catgut. Careful hemostasis and gentle treatment of the tissues are required.

As much as possible of the raw surface left by the hemilaryngectomy must now be covered with mucous membrane. The above mentioned flap is brought downward and inward. Care should be taken to avoid tension in stitching the flap in its new position with fine iodized gut. The tracheal packing is now removed and the trachea cleared of blood clot down to the cannula. A long strip of vaselined gauze, thoroughly impregnated with bismuth, is packed into the trachea. The end of the strip is brought out at the lower end of the laryngeal incision. The larynx is packed loosely with a strip of iodoform gauze, the end of which is brought out at the same point as the tracheal packing. The gauze in the pharynx is removed through the mouth, and the feeding tube is pushed through the nose and directed into the esophagus.

The mesial edge of the pretracheal muscles is now united to the mucous membrane of the healthy side of the larynx along its anterior median aspect, using fine iodized gut. The skin edges are united with silkworm gut. The wound is left open at its lower angle, wide enough to give drainage soon for the gauze ends. Two provisional stitches are placed here, to be tied after these strips are removed, thus entirely closing the larynx. A sheet of rubber tissue, covered on both sides with gauze, is glued by its edges with collodion to the neck, across the bridge of skin separating the two wounds. This attachment should extend laterally one-half way around the neck to keep the drainage of the laryngeal wound from the tracheal cannula.

As after-treatment Dr. Mackenty prescribed a diet of oatmeal, thoroughly cooked and strained, mixed with milk, milk-sugar or raw egg, and occasionally with melted butter. Six ounces of this mixture is forced through the esophageal tube with a piston syringe every four hours, followed by four ounces of water.

To clear the trachea of secretion, suction is used as needed. A small catheter is passed down to the bifurcation, and about ten pounds of negative pressure is applied. Dressings should be changed evey few hours. The laryngeal drain should be kept in as long as the serous flow continues. The tracheal plug may remain from three to five days. The tracheal cannula should be removed just as soon as the new larynx is open for comfortable breathing.

DISCUSSION.

DR. JOHN F. BARNHILL, Indianapolis, thought the method described would undoubtedly prove advantageous in the very early cases in which it was possible to make a diagnosis. The new feature of the method was the forming of a cavity lined by mucous membrane. This, of course, would presuppose the presence of good membrane to be used for this purpose. For this reason the method was applicable only in cases in which the disease was not very extensive. If Dr. Mackenty had not had six recoveries in his six cases, he would have been open to the criticism that he should have done laryngectomy. Semon, Butlin, and others who had done a great deal of this work, had reported better results with laryngectomy than with hemilaryngectomy. With the modern methods for keeping the secretions out of the larynx it was possible to do more than might be done otherwise. Crile, who had done a good deal of this work, advised doing two operations, the first being a low tracheotomy, for the purpose of getting the patient accustomed to the tube. In Dr. Mackenty's method that was not necessary, as he succeeded in keeping the secretions out of the trachea, so that there was no danger of the fluid dissecting its way down into the mediastinum, as happened in many cases.

He asked if there had been stricture in any of Dr. Mackenty's cases. In case there was not enough mucous membrane to cover the wound, there might be stricture after removal of the tube.

Dr. Mackenty, in closing the discussion, said one case had refused total laryngectomy. He had not had any postoperative strictures. He did not think the Semon operation applicable in any of his cases. He got enough mucous membrane to cover one-quarter or one-third of the wound in most of the cases, as he had stated in the description of the operation. He was opposed to the two-stage operation. It was not done abroad, and he did not see why it had to be done here. In going in for the second operation it was necessary to go through a septic field. In total laryngectomy he believed in the complete operation—i. e., with the gland bearing tissues of the neck. In one case there was a fairly tight point below the larynx, because he had to do more than a hemilaryngectomy. Moreover, the patient had a distorted trachea from the enlarged thyroid. Answering the question about the perichondrium, he had always taken the mucous membrane without perichondrium.

Cleft Palate and Hare Lip.

Dr. Owen Smith: Careful study of the operative technic for cleft palate reveals three distinct methods with minor modifications:

1. Elevation or separation of the soft tissues of the palate, and suturing the denuded edges in the middle line, as advocated by Langenbeck and Warren.
2. Flap elevation, reflection or rotation, as advised by Lane.
3. Forcible closing together of the cleft by lateral pressure and retaining the parts in place by heavy metal sutures, as suggested by Brophy.

Having operated upon more than two hundred cases, and having carefully tried all three plans, the essayist unreservedly commended the second method, suggested by Lane. The most important reason for an early operation is that a large number of these patients die before they reach the age when they would be ready for operation by the older methods. His own experience leads him to the belief that fifty or sixty per cent die of inanition within the first six or eight months of infancy, if the cleft is not closed. The mortality from early operation by the older surgeons was so great that they were forced to abandon it. In contradistinction, Lane and Brophy did not have any large percentage of deaths from their work, and in his own series by their methods he had not had a fatality. The sooner after birth the operation is performed, the less hemorrhage, while the shock to the nervous system will be very slight. The muscles of the throat after the early operation begin their activity at once and do not have time to atrophy. This atrophy takes place where operation is delayed, and the eustachian tube suffers from this disuse of the muscles. The early formation of a fibrous band between the edges of the cleft by contraction begins at once to mould the twisted face into its proper position. Before the teeth erupt an opportunity is offered to get a wider flap by extending the incision outside of the alveolus. The immediate repair of the lip adds another factor in restoring the palatal processes to their normal place. Lane's operation is designed to bridge over the cleft by means of mucoperiosteal flaps from the bony palate in front of the mucous membrane and submucous tissue from the muscular or soft palate behind. Flaps properly formed, when placed in position over the cleft, present their raw surfaces together, one overlapping the other. Fine silk sutures are used, and when tied they unite the flaps in this way and form a new, strong and complete palate. It has been the essayist's practice, in order to hasten the healing, to curette the ulcerated free border of the septum, often found at birth, and to dissect up the mucous membrane on both sides, until material enough is obtained for a flap, which is pulled down and sutured over the free edge of the curetted septum. It is never safe to operate while the ulcer is present. For some time he has thought it wiser to divide the operation into two sittings, particularly if the cleft is very wide. The technic is described in detail.

The hare lip operation employed by the essayist is designated as the lip splitting method. In this operation there is no sacrifice of tissue, and the lip may be operated upon as many times as necessary until the desired effect is obtained.

DISCUSSION.

Dr. John Edmund Mackenty, New York City, said Dr. Smith had improved upon the Lane operation. The objection he had had to the Lane operation was that it called for the splitting of the soft palate into two parts, more or less completely ruining it as a functionating organ. He would try out Dr. Smith's method in future. He had never done a hare lip operation without undermining the tissues of the lip so as to relieve tension at the edges. It was not quite clear to him that tension would be relieved by Dr. Smith's method. It had not been his experience that such a large percentage of cleft palate patients died. He would like to hear more about the speech results in the Smith method, and it was to be hoped that Dr. Smith, in future papers, would tell of the speech results in his cases. He had used an obturator in the mouth to prevent tongue pressure, and had obtained better results when he did this. Most of his operations had been done by a modification of the Langenbeck method.

Dr. Mackenty presented a model of his obturator.

Dr. Truman W. Brophy, Chicago, said there were so many varieties of defect that it was difficult to discuss a paper on this subject. He classified hare lip under thirteen forms, and cleft palate under fifteen forms. He presented charts showing illustrations of the various forms. In discussing cleft palate it was necessary to consider the different kinds, because there were nearly as many different kinds of treatment as there were congenital defects. The object in operating upon the palate was two-fold: First, to overcome the congenital deformity, to restore the parts just as nearly as possible to normality; second, to correct the condition so as to enable the patient to speak properly when the age of speech was reached. The surgeon who failed to accomplish this failed to attain success. A cleft palate was like an open wound. A surgeon would not make a patient wait before closing an ordinary open wound, and yet many surgeons made patients wait for years before closing a cleft palate.

Lantern slide illustrations of different forms of defect were shown, and a description of his technic for each, with the reasons for the same, were given.

Dr. George L. Richards, Fall River, said, in justice to Dr. Smith's method, that he had a case under observation which corresponded to Dr. Brophy's form XIII. The patient had a good lip, a good palate, and was developing a very good voice. One would hardly know the patient was not a normal individual.

Dr. Smith, in closing the discussion, did not dispute Dr. Brophy's ability to do anything with wires. In order to pass a verdict upon the two operations, one should try both. Every case in which he had employed the Brophy operation had been an absolute failure. Dr. Brophy obtained the desired object by one method, he obtained it by another. He had no desire to criticise the Brophy operation. In the method described there was an immediate formation of fibrous tissue which drew the parts together, if taken early enough. One could obtain any desired length of palate by taking a little care in placing the flaps before beginning the sutures.

Some Observations on Turning Nystagmus.

Dr. George W. Mackenzie's investigations, covering a period of eight years (1906 to 1914), have resulted in findings contradictory to those of Bárány on after-turning nystagmus. He believes the observations of the latter have been faulty because of inaccurate technic, and also that the wide discrepancies in the duration of after-turning nystagmus in normal individuals, claimed by Bárány, do not exist. Hence the author concludes that the hearing test is a valuable one in the diagnosis of labyrinthine conditions.

The author examined one hundred and seventeen cases in all, including nine normal individuals. The list included nearly all of the varieties of ear diseases to be met with in the average practice. They differed from Bárány's in the following particulars:

In normals, Bárány found the average duration of horizontal after-nystagmus to the right after ten turns to the left was forty-one seconds. The author found that the average duration of horizontal after-nystagmus to the right after ten turns to the left was a fraction of a second less than twenty-four seconds. Bárány found that the average duration of horizontal after-nystagmus to the left after ten turns to the right was thirty-nine seconds, and the author found that it was only twenty-four seconds. Bárány: The maximum duration for horizontal after-nystagmus to the right after ten turns to the left was ninety-eight seconds; the author: twenty-nine seconds. Bárány: The maximum duration for horizontal after nystagmus to the left after ten turns to the right was one hundred and twenty seconds; the author: thirty-one seconds.

Bárány mentions one case with no nystagmus after ten turns, and uses this as his minimum for after-turning nystagmus. The author observed two cases with apparently no nystagmus, but upon repeating the test and turning the patient at a more rapid speed, and examining carefully, he was able to note and time the nystagmus, when it was found to be of relatively short excursions and lasted the usual length of time. Bárány observed horizontal after-nystagmus averaging forty seconds in sixty individuals examined on different days. The maximum was eighty seconds; minimum, twenty-five seconds. One-fifth of the cases showed consistently the same findings; in other words, four-fifths showed variable findings. The author examined a smaller number of such cases, and found the average after ten turns for horizontal after-nystagmus to equal about twenty-four seconds. The greatest variations were but a few seconds, and these could readily have been explained on the ground of faulty observations. In a number of cases in which Bárány tried both rapid and slow turnings, he has found that after ten rapid turns the nystagmus lasted from eighteen to nineteen seconds, while after slow turns it lasted from twenty-six to thirty seconds. In one-half the cases he found the rapid turns produced a longer after-nystagmus than slower turns. The author tried patients with both rapid and slow turnings, with the result that the duration is apparently less after after slow than after rapid turns and consistently so. However, most careful tests will show them to be about equal in duration. The difference is more apparent than real, for the reason that after slow turning the nystagmus is less intensive than after rapid turning, hence less noticeable and more difficult to determine the moment of cessation. Bárány claims that with a great number of persons the horizontal nystagmus after twenty, thirty, forty, fifty and sixty turns was the same as after ten turns, adding that it appears that in the average ten turns show the Hochster Werte (highest worth, value or rate), while with a greater or less number of turns the amount diminishes. The author admits that he is not in the position to match Bárány in material, but he performed some autoexperiments of this kind, and found the nystagmus to increase consistently in duration between ten and twenty turns. He found, furthermore, less variations after twenty than after ten turns. Bárány claims that the amount of after-turning nystagmus after twenty turns is about equal to that after five turns. The author never found this to be true in a single case in his experience. Bárány claims that after twenty or more turns there appears not infrequently, a nach-nach nystagmus (after-after-nystagmus) in the direction opposite to the after-nystagmus, lasting sometimes a minute. The author has never seen an after-after-nystagmus in many hundred examinations, including those in his list. He does not believe it exists, and will not believe it until it is shown to him. Concerning dancers, Bárány claims that with those who habitually turn to the right the duration of the horizontal turning nystagmus to the left falls considerably shorter than that to the right. The author was fortunate enough to obtain a whirling dervisher with the stage name of Hash Hash, who was a left turner. The author examined him on many occasions, and found that his duration of nystagmus was consistently the same on several occasions. The figures were reduced for both horizontal and rotatory nystagmus to both sides, and more so to the right. This one case supports the claims of Bárány.

The author further pointed out that Bárány, in his efforts to find a relationship between the semicircular canals and the eye muscles, neglected a certain caution that he himself warns against when he advises the use of opaque glasses in studying horizontal after-turning nystagmus. The author attributed Bárány's irregular findings, as compared to his own, as due to his technic, which the author believed faulty, rather than to actual irregularities in the behavior of the reflexes.

In Bárány's "Table for Differential Diagnosis of Mentioned Diseases," page 276 of the Proceedings of the Ninth International Otological Congress, he indicated with arrows the direction of spontaneous nystagmus in the column, suppuration of the right labyrinth since twenty-one days, and elsewhere states that the patient has spontaneous nystagmus to the right as well as to the left. The author remarked that this nystagmus to the right, when looking to the right, is not in the least way connected with the patient's labyrinthine condition, and that Bárány has included it among other findings in labyrinthine suppuration only supports the author's previous contention that Bárány lays more stress upon the side positions than the straightforward position of the visual axis. In Bárány's table there is no mention made of his having noted the spontaneous nystagmus that occurs when looking straight ahead, while the author considered no other nystagmus of any value from the standpoint of the semicircular canals. When the intensity of the nystagmus does not balance in opposite directions, the author accepts it as pathologic, and the problem then is the isolation of the particular muscle or muscles involved.

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLOGY.

Meeting of November 12, 1915.

DR. CORNELIUS D. VAN WAGENEN, CHAIRMAN, PRESIDING.

Purulent Leptomeningitis of Otitic Origin With Recovery.

DR. ISIDORE FRIESNER presented a patient who had made a spontaneous recovery from purulent leptomeningitis of otitic origin. He said that Preysing, in his essay on meningitis, stated that if one reviewed the case reports of healed meningitis, one was almost ready to believe that we were making rapid strides toward the conquest of this most formidable otologic complication. Quite a number of cases of meningitis had been reported during recent years in which, by the exhibition of one or another form of therapy, by either surgical or medical means, a cure had been effected. And yet the total of cured cases was so pitifully small that grave doubt still existed in the minds of many able observers as to whether diffuse purulent leptomeningitis ever ended in recovery. It was possible that the following case report might in some small degree help to clear up this question. It differed in one essential from most of the other cases of healed meningitis reported in that there was nothing to offer constructively. This case, which there was justification for considering a diffuse purulent leptomeningitis, recovered spontaneously. The young man, J. B., sixteen years old, went to Dr. Phillips' clinic at the Manhattan Eye, Ear and Throat Hospital on June 13, 1915. His right ear had been discharging since he was ten months old. After operation it was learned that except for this discharge his ear had never troubled him until several days before entrance into the hospital. The patient was then seized with sudden pain in the ear, accompanied by headache, fever and vomiting. Functional tests showed his labyrinth active and normal. On June 15th Dr. Friesner had assisted Dr. Shook, who was then house surgeon of the Ear Division, to perform a radical mastoidectomy. The mastoid was found sclerosed;

there was neither pus nor cholesteatoma; the middle ear contained a little granulation tissue. The dura of the middle fossa was exposed and it appeared normal. Immediately following the operation the boy's temperature rose to 105°, accompanied by intense headache and vomiting. During the succeeding eleven days his temperature ranged between 102° and 105.5°, he vomited frequently, was delirious, his cervical vertebræ were tender, the neck was rigid and head retracted. There was a marked Kernig in both legs and an herpetic eruption about the lips and the opposite ear. On the day following operation lumbar puncture showed very turbid cerebrospinal fluid; microscopically it contained pus cells and fairly numerous short chain streptococci, and culture from the spinal fluid showed streptococci. The labyrinth was reexamined several times and found active. Permission for exploration was withheld until, believing the boy's condition was so desperate that the condition was a meningitis which would prove rapdily fatal, it was thought unwise to interfere. The fever persisted for forty-one days, and during the first three weeks he was frequently delirious. The headaches became less severe after the first two weeks. The rigidity of the neck persisted for a month, and the Kernig was positive even after his temperature was normal. Late in the disease, severe girdle pains and pains in the arms and legs were observed; these were believed to be referred pains due to irritation of the posterior special roots. Even after improvement was evident it was doubted that recovery could ensue, as this was believed to be one of those intermittent meningitides described by Brieger, and it was expected that the boy, who appeared on the road to recovery, would suddenly become unconscious and die within a few hours. The radical cavity has healed nicely and is perfectly dry. Healing is present to a very small degree and the caloric is weakly positive.

DISCUSSION.

Dr. H. L. Lynah said that he had had a case of otitic meningitis resulting from scarlet fever. The patient was operated on and injected with fifty cubic centimeters antistreptococcus serum. An acute urticarial rash developed, which was regarded as a favorable prognostic sign; this proved correct, as recovery followed.

Dr. Wells P. Eagleton said that he had been particularly interested in Dr. Friesner's paper, as it dealt with a number of points which he himself had long been interested in. One point to which Dr. Friesner had referred he had never seen mentioned in print, but had observed it once and had written about it, but he had then lost his nerve and failed to publish it, namely, that thrombosis is not prior to cerebellar abscess, but secondary to it. Several years ago he had a patient with a cerebellar abscess. At that time all the surgeons were following the teachings of McEwen, who said that cerebellar abscess was generally caused by sinus thrombosis. This patient had no symptoms of sinus thrombosis. He became comatose. Finally the cerebellum was opened and a large abscess was found, which should have been opened two or three weeks earlier. On cutting through into the sinus the latter was found to be empty. The man died some time later, and postmortem showed no trouble with the sinus. The fact of finding an empty sinus with a big cerebellar abscess made a great impression on him, and on thinking it over he came to the conclusion that with a greatly increased cranial pressure the first thing to give way would be the large venous trunks, and if there was a superficial cerebellar abscess, it seemed possible that it might press out the blood, as we now know that a tumor does press the blood out of the sinuses to make room for itself. The infection being in proximity with an empty sinus might tend to cause a secondary infection of its walls. He thought that might be the explanation, though he had never heard such a case reported.

Otitis Media Purulenta Chronica—Mastoidectomy—Meningitis With Unusual Focal Symptoms—Postmortem Findings.

Dr. C. D. Van Wagenen read this case report of a man, Peter P., age twenty years, single, who was admitted July 3, 1915, to the Manhattan Eye, Ear and Throat Hospital with a diagnosis of otitis media purulenta chronica, right and left, acute mastoiditis right; emergency operation. This case, occupying the bed adjoining that of the very unusual case of purulent meningitis of otitic origin which recovered spontaneously, presented here tonight by Dr. Isidore Friesner, was reported for its contrasting meningitic picture and its illuminating and valuable autopsy. The family history was negative, as was the previous history, in the main; the patient, however, supplied the important fact that there had been an intermittent discharge from both ears since early childhood. More recently, in 1913, this patient had been confined to the ward by a right otitis media purulenta acuta, which had apparently completely resolved under myringotomy, rest in bed and appropriate local treatment; a residual condition of the left middle ear was observed at this time; the patient then entirely disappeared from observation until has last admission to the hospital, when he gave a history of having had a discharge from both ears for fourteen weeks, with headache gradually increasing in severity in both the frontal and occipital regions, but unaccompanied at any time by dizziness, nausea or vomiting. Upon receiving the report, about the noon hour, from the house surgeon, Dr. Brownlee, that there were marked signs of a right mastoid involvement, and that the patient seemed a very sick man with temperature of 101°, respirations 24 and pulse 106, preparation for operation was ordered. The operation, a simple mastoidectomy, was performed by Dr. Van Wagenen on the afternoon of the day of admission. The whole area of the cellular mastoid was found in a necrotic condition, bathed in foul smelling pus from which gas, under pressure, immediately issued. An extensive exposure of the lateral sinus showed it to be thickly and uniformly covered by a layer of dirty gray granulations, resilient and pulsating under the palpating finger. The wound was left wide open, loosely packed with sterile gauze, a heavy wet saline dressing was applied and the patient was returned to the ward. As the left ear was found to be discharging moderately, hydrogen peroxid, followed by a saline douche, was ordered q.—3—h. During the night of July 4th the temperature rose to 105° F., with respirations 24 and pulse 100, but sank to 104° in the early morning, being possibly influenced by an alcohol sponge. Dr. James G. Dwyer, the pathologist of the hospital, reported that the smear taken from the operating field showed a mixed infection with the pneumococcus mucosus predominating. The patient was resting quietly. On July 5th Dr. Dwyer reported the blood culture as negative after twenty-four hours. As a matter of fact it was negative throughout. The temperature, pulse and respiration remained high and the patient was somewhat restless. Systematic treatment of the left ear was producing results. Beginning with

July 6th the patient grew progressively worse. There was a conjugate strabismus. The fundi were found to be normal except for a slight engorgement of vessels. There was a rather profuse hemorrhage from the nose. The patient began complaining again of pain in the head; one observer thought that this pain could be sufficiently well localized in the left mastoid to permit of a diagnosis of mastoiditis, especially when taken with the discharge and appearance in the middle ear. The appearance of a left facial paresis during the closing hours of this day seemed to strengthen this belief, but the increasing prominence of the meningitic picture overshadowed all else. The next day, July 7th, paresis of the left arm and leg appeared and the restlessness increased at times to an active delirium. Dr. Dwyer examined about thirty cubic centimeters of turbid fluid from a lumbar puncture in two tubes; there was considerable precipitate of pus made up of red and white cells and pus cells. Sugar reducing substance was absent; lactic acid marked. Smear was negative bacterially; culture after twenty-four hours was negative and finally negative. Death ensued, with a gradually falling temperature, at 9:50 p. m., July 8th.

At autopsy Dr. Dwyer found general leptomeningitis with a large collection of pus between the cerebrum and the cerebellum, especially on the left side, simulating abscess, and thus giving focal symptoms; pus in large amount over the cerebral cortex, especially following the longitudinal sinus; considerable necrosis of the anterior end of the right frontal lobe, not at the bottom of the lobe, but in the ascending frontal surface; abscess of the right frontal lobe was not yet broken down into pus. The sinuses were all open and clear. Left mastoid very pneumatic and clean. Paresis of left arm and leg was due to cerebral lesion at fissure of Rolando, and left facial was due to pressure there or at the point of collection in the tentorium. Culture showed pneumococcus mucosus.

DISCUSSION.

Dr. M. Nicoll, Jr., said that he would like to hear Dr. Dwyer, who made the examination of the lumbar puncture, tell something more about it, as sterility of the turbid fluid was not a usual feature.

Dr. J. G. Dwyer said that quite often in a smear the organisms were found without a direct growth, but that in this lumbar puncture the culture was negative.

Dr. Meirhof showed two X-ray plates. One of a man of thirty-five years, with a history which showed he had been ill five weeks, complaining of pain in his ear, in which incision of the drum had been made; temperature of 103° and headache. Examination of the ear showed no local symptoms, no exudate at the time, no swelling of the canal, no tenderness over the mastoid on deep pressure. Ear tests showed practically a loss of hearing. Blood culture was negative. Leucocytosis was high. The mastoid looked cloudy in the radiograph, and the radiologist thought there were some changes in the sinus. The X-ray picture was the means of giving some idea of the condition present. There was distinct contrast of the normal to the abnormal side. Lumbar puncture was made, and the fluid came away very cloudy. At operation pure pus fell out of the mastoid. The sinus looked blue.

The other case was a boy of fifteen years. In August, while at camp, he had a swelling behind the left ear which he thought was the result of a spider's sting. Three months later he had pain over the left mastoid which was very severe. Six years before he had had otitis. There was no exudate; he could hear a watch tick at fifteen inches; an X-ray was made which showed a normal mastoid on the affected side. Blood count was normal. He continued this way for several weeks, and it was not believed that he had any disease, but that the condition was one of hysteria or neurasthenia. The father finally admitted he had struck the boy over the ear rather violently in anger.

Dr. Harold Hays said that he wished Dr. Dixon were present to interpret the first picture, as from the appearance of the plate he did not consider the mastoid very extensively involved, though of course it must have been, as there was pus present. He asked if there was exposure of the dura, and suggested the possibility that the case was one of meningitis in which the mastoid had become involved.

Dr. Ernst Danziger said that the case as reported struck him as an absolutely clearly defined one. The headache, the fever, and the sudden development of complete deafness of the affected ear was a typical clinical picture of an acute purulent labyrinthitis. He regretted that there had been no mention of nystagmus or tubercular reaction. The thrombosis was only of secondary importance.

Dr. H. B. Blackwell said that two years ago he reported two cases in which there were no objective signs of mastoiditis. They were X-rayed, and the findings were different in the two cases. The X-ray findings in one showed the mastoid was involved. In the other, where the X-ray was negative, meningitis developed seventy-two hours after the picture was taken, and the patient died. The mastoid was opened and showed that it was very slightly diseased. The point to be emphasized in X-ray diagnoses is that it is impossible for the X-ray to show the grave character of the mastoiditis unless the mastoid has become pretty well diseased. Sometimes the X-ray will show a normal condition which may result in meningitis, as in his case. Diagnosis in these cases has to be made on the more or less indefinite clinical symptoms which the patient presents —the malaise, history, headache and restlessness, the slight temperature which they sometimes have, 102.4°, and lastly the X-ray findings were to be taken. If the X-ray was negative, the condition might not be very grave, but if positive, the diagnosis was confirmed.

Micropathology of Brain Abscess.

Dr. Wells P. Eagleton read a paper on the micropathology of two cases of brain abscess, illustrated by lantern slides.

DISCUSSION.

Dr. H. S. Martland showed some specimens and said that the case of encapsulated abscess that Dr. Eagleton had referred to was from a case that he had autopsied. The brain specimen, showing the abscess, he passed around for examination. The patient had a dural endothelioma situated in the right cerebellopontine angle. Four months before his death he was trephined over the right cerebral hemisphere near the median line, and a puncture of the right lateral ventricle was made. At autopsy the right cerebral hemisphere presented a round abscess cavity, four to five centimeters in diameter, containing foul, greenish pus, in which a few streptococci were found. The walls of the abscess cavity were four to five centimeters in thickness, and on microscopic examination presented three distinct layers; the inner was composed of disintegrating and sloughing brain tissue, fibrin and bacteria infiltrated with polynuclears; the middle was composed of connective tissue with extensive fibroblast proliferation, due to stimulation from the presence of blood and fibrin; and the outer was made up of edematous brain tissue containing a protective wall of endothelial leucocytes. There was no infection of the lateral ventricle, the inner wall of the abscess approaching very close, however, to the ependyma of the right lateral ventricle. From the outer part of the wall of the abscess to the brain surface was an area of old cerebral softening, the remains of the track of the needle during puncture, proving that the infection was carried into the brain at the time of the needle puncture. A second specimen that Dr. Martland passed around for examination showed two small abscesses of the brain due to pneumococcus infection, and occurring as a complication of bronchiectasis.

Dr. Gutman said that he believed brain abscess of otitic origin to be always an extension of infection, and asked Dr. Eagleton to explain how the thrombosis phlebitis, coming down from the brain, could cause brain abscess, and also asked how he could explain the length of time it took for the capsule to form.

Dr. M. Nicoll, Jr., said that he wished to thank Dr. Eagleton for his splendid exposition of the pathology of brain abscess, and asked if Dr. Eagleton had seen the thrombosis of the vein during the operation or at postmortem.

Dr. Eagleton replied that he thought Ballance had misled many about looking for the stalk. That procedure had hindered progress. The reason for the absence of the stalk was very simple and was now well recognized. The cortex of the brain had a very complete vascular supply, while the interior of the brain had a very poor vascular supply. Infection reached the cortex from the infected area in the ear, but the supply of the cortex was so great that the local death from the nutritional disturbance did not occur. The thrombosed vessel, when it once reached the intramedullary portions, reached the portion where the brain was dependent for its nutrition on the small venous trunk. All abscesses extended inward for the same reason. They never extended outward, as they went toward the ventricles, not outward. He had seen surgeons open the head and, not finding the abscess, leave the dura open, saying that would give the abscess a chance to come out. Because of the local death inward, all the line of resistance was toward the ventricle and not toward the cortex. The otologists as a whole were to be congratulated upon the advanced work they were accomplishing in the physiology of the cerebellum. The neurologists had contributed but little surgically, but the otologists had been dealing with the problem from the surgical standpoint and had learned much about the very delicate mechanism of the neurology of the cerebellum.

Aural Complications in Exanthemata.

Dr. Henry L. Lynah read an article on the aural complications in exanthemata. He said that the percentage of otitic complications were governed by the degree of the local throat lesion. They were caused by the extension of membrane through the eustachian tube. The scarlet fever percentage was high in the nasopharyngeal type. There was early and late infection. The aural lesion was often obscured. In measles the percentage was high in the catarrhal stage with fulminating aural complications. Age, pain, temperature, heat and cold were factors. There was a question if latent complications were due to aural lesions. There were anomalous types. There was sinus thrombosis with and without aural involvement. The complications were sometimes brain abscess, labyrinthitis and meningitis. Preventive measures were removal of tonsils and adenoids; nose and throat irrigations; injection of antistreptococcus serum. Radical or conservative treatment had effect on the hearing apparatus and the mortality of the case.

DISCUSSION.

Dr. M. Nicoll, Jr., said that Dr. Lynah's paper seemed to cover the ground very well. In reference to lavage being likely to cause middle ear infection as well as the Casselberry position, it was easy to conceive that the latter might induce infection through the posterior nares, but that lavage could not be held responsible for infection, and those two should be separated. But neither of those two methods are used now. It had always been interesting why diphtheria did not cause middle ear lesions; not the diphtheria bacillus alone, but the streptococcus found with it; why was there not a suppurative condition of the middle ear, as in scarlet fever? Regarding irrigations, he had been so opposed in his theory that he hesitated to reaffirm it, but he believed that irrigation, done properly, without roughness or undue pressure, in certain cases with a great deal of discharge, was the proper procedure; that middle ear disease did not follow any more readily than if it was not done; that it was rational treatment and could not do any harm. Where the heart was weak and the child struggled, it might be a poor procedure, but there were certian cases where it ought to be done. In regard to serum locally, he was more in favor of that than intravenously; it had some clearing up value and was soothing and agreeable to the patient.

Dr. G. Pisek said he thought the section was more especially interested in the aural complications. It was interesting to look back into history and appreciate that the aural discharges were the last stand of the adherents of the humoral pathology. Dr. Lynah gave the statistics of complications in the infectious diseases and they were low, but that at certain seasons of the year and at different periods the infection had been of a more virulent type, and then more complications appeared. In the summer particularly the infectious diseases are few in number; but at all times of the year, in modern, properly equipped hospitals, the complications are becoming more rare. The interns, for example, are equipped with an auroscope, and they make it a part of their routine duty to make careful examinations of the ear. With such a routine the more important complications are apt to be forestalled. If there was marked nasal involvement, there was more apt to be aural involvement, and then the ears were more carefully watched. The pediatrist was often called in to assist the otologist, from the differential standpoint, in cases of suspected mastoiditis where the question of pneumonia came up. If the child had recently had a mastoidectomy, bulb involvement or pneumonia were probable. Blood culture was often necessary, though it should be recognized that only under certain conditions was the blood culture reliable. Unless, for example, the blood culture was taken just before the rise in temperature, negative blood culture might be reported. The pediatrist and aurist needed to take counsel together in these complicated infections, and they should insist on blood culture being made and taken at the proper time. The mortality could be reduced if early diagnosis were made.

Dr. Meirhof said that Dr. Lynah's paper seemed a comprehensive one, but that there were a few points he wished to touch upon. The question of the hearing of these children interested him very much, as an examination of two hundred and eighty mutes had shown their condition to be due to suppurative otitis from scarlet fever, diphtheria, etc., and made him believe that it is not known what the final outcome of these cases will be; the question was, was the deafness of these mutes due to indifference in their treatment, or would appropriate treatment have saved their hearing? There was a very rapid destructive process going on. The use of the syringe tended to keep up the formation of granular tissue.

Dr. Opdyke said he had seen only one case where there was a distinctly diphtheritic membrane in any case of mastoiditis, and it was a very virulent case. Notwithstanding the injection of diphtheria antitoxin and thorough evacuation of each sequestrum, the patient succumbed. There was nasal and aural involvement, and both mastoids were diphtheritically affected. Speaking of measles, he was called into see a girl who was prostrated with whooping cough, which was followed by diphtheria and then measles. Both ears were affected and a mastoidectomy was done on one side. There was no pus in the mastoid antrum. Lumbar puncture was negative. The rash of the measles did not come out; the temperature was 105°; on the seventh day after the mastoid operation the rash came out and the child succumbed. He wondered if that retained rash was the cause of death, or was it meningo-encephalitis.

Dr. John A. Robinson said that four years ago he saw a patient who complained of sore throat for two days and stuffiness in her left ear. Temperature was 102°. She looked ill. The mucous membrane of the oropharynx and faucial pillars were deeply congested. The uvula was edematous. The glands at the angle of the jaw were enlarged and the membrane was congested. Owing to the thickness of the parts, posterior rhinoscopy was unsatisfactory. With the nasopharyngoscope of Holmes the pharyngeal opening of the left tube was seen to be coated with a grayish membrane. A culture made from a smear showed this bacteriologically to contain Klebs-Loeffler bacilli. No other membrane was visible. The exudate was so characteristic that immediate administration of antitoxin was urged to the family physician in a neighboring suburb, which he did the following evening. Twenty-four hours later, membrane appeared on both pillars and on the left side of the pharyngeal wall. The exudate did not extend into the nose. The patient developed a mild acute otitis which subsided without myringotomy. She received four doses of ten thousand units. As sequelæ she has a markedly narrow tube, a low whisper distance of six feet, and a low pitched tinnitus.

Dr. J. J. McCoy said that he was impressed by Dr. Lynah's paper with the low percentage of involvement of the labyrinth, and thought the secret of success in these cases was the early treatment. There were many cass of severe sepsis in scarlet fever in which the kidney was involved and the child had single or double mastoiditis, when there was a question whether immediate operation should be done, or the antrum simply drained, which it would seem advisable to do at that time, later completing the mastoid.

Dr. Lynah said he agreed that in such cases the serious operation should be delayed, and that it was better to merely do myringotomy at first. If the drum membrane sloughed out, it made no difference in the hearing, as that was injured anyway.

The meeting was closed after Dr. John A. Robinson had been nominated as Chairman and Dr. Harold Hays as Secretary for the coming year.

NEW YORK OTOLOGICAL SOCIETY.

Meeting of January, 1916.

A Case of Congenital Bilateral Microtia—Presented Five Years After Operation.

DR. JOHN R. PAGE: The patient, a small boy, seven years of age, was first seen by Dr. Page when he was six weeks old, and was observed from time to time for two years before the right ear was operated on. The left ear was operated on one year later.

A certain degree of hearing had been demonstrated by the child's attention being attracted by whistles, etc. X-ray suggested the presence of a tympanum and ossicles on both sides. Dr. Page said that the patient's family were very enthusiastic in their praise of the result, as the boy had learned to talk, and the mother assured him that she had no more difficulty in talking with him than she had with the other children. When spoken to from another room he responded quickly.

The case was reported in detail, and was printed in the Transactions of the American Otological Society in 1914. There was found on both sides a total bony atresia and a contracted tympanum with rudimentary ossicles. There was now a radical cavity on both sides which admitted a speculum of medium size on one side and a large speculum on the other. Considerable cicatricial contraction had occurred, but allowance had been made for it at the time of operation.

Dr. Page said that in none of the textbooks had he been able to find any encouragement to operate on such a case. In spite of the authorities, Dr. Page said he believed that in the majority of such cases, if the child was perfectly normal otherwise, the hearing could be improved by proper operative interference because of the fact that the essential organ of hearing is developed from an entirely different origin than that of the external auditory canal and auricle. Dr. Berens had had a similar case, and had obtained a good result from operation.

It has been suggested as one of the frequent causes of failure in these cases that the anterior wall of the bony canal is invariably absent, and the articulation of the jaw rests far back, practically against the mastoid process. It has been suggested that ignorance of this fact has been the cause of so many operative failures. The operator, in attempting to follow what he supposed to be the line of the bony canal, encounters the articulation of the jaw or the facial nerve. The best procedure is to enter far back on the mastoid, expose the sinus and middle temporal fossa, if necessary, and get into the antrum and use the horizontal semicircular canal as a landmark; work from the aditus out and widen up a large cavity, exposing well the internal wall of the tympanum, and treat it as a radical cavity with the skin grafts, etc.

DISCUSSION.

DR. THOMAS J. HARRIS said that he wanted to ask Dr. Page whether a good many of these cases which had been operated on showed complete absence of all vestige of the middle ear. The speaker said it was his impression that such was the case in these patients. The explanation that the middle ear had never been entered because of a faulty channel might be the correct one, but after the most extensive operation they could find nothing at all.

DR. WENDELL C. PHILLIPS said that he had given some study to these cases, and had concluded the crucial point was to decide whether the patient had hearing. It was proper to operate if the patient could hear. Most of the cases under his observation had no hearing, and it seemed to him an unwise procedure to make a canal where there was none. The speaker stated that he had a lifelong opportunity to study one case in a member of his own family, who had one rudimentary auricle, but seemed to have no hearing at all. He had no external auditory canal, but a fairly well shaped ear, which had remained infantile during his whole life, and a well formed eustachian tube. Inflation seemed to simply open up the tube. The speaker said this was the case he had illustrated in his textbook.

Dr. John R. Page, in reply to Dr. Phillips, said he thought, contrary to what Dr. Phillips had said, that hearing could be demonstrated in practically all of these cases. That investigation had shown the deformity had never occurred in both the internal and the external ear at the same time except in cases of markedly undeveloped and deformed infants and nonviable embryos. He did not think that operation was warranted in the unilateral case where there was good hearing in one ear. After hearing had been demonstrated in this case, X-ray pictures were taken and the child was operated on when he was two years old. One year later the other ear was operated on.

Dr. T. Passmore Berens said he had such a case as the one to which Dr. Page had referred. He had forgotten the exact age of the patient, but thought the child was about three and one-half or perhaps four years of age; an institution child, a foundling. On one side there was only a rudimentary tragus, no external and no bony canal. On the other side there was a little miniature auricle, not much bigger than the nail of the forefinger, with no meatus. Her attention could be attracted by any kind of noise. If someone stamped on the floor with the foot she would look around.

Dr. Berens said he operated on one side on the ground that the deformity was so great that it would do her no harm, even if the hearing should not be improved. He selected the most rudimentary side with the small bit of a tragus, and cut down to where he supposed the canal might have been and came to the capsule of the jaw. This was not opened. He followed from the temporal ridge downward and inward, hugging the internal table covering the middle fossa, and finally fell into the attic, and from the attic went into the antrum and found there a rudimentary incus but no malleus. The incus came out in the operation, and one could see the stapes, the nerve and the external semicircular canal. On that side Dr. Berens performed a saucer operation and scooped out all the bone he possibly could, and made as flat a wound as he was able. It healed nicely. The patient was kept in the hospital for a couple of months. She commenced to mumble with the other children and began to hear. Dr. Berens operated on the other side, and there also failed to find any bony canal, but found a meatus and the ossicles, which healed open. She was kept in the hospital for six months, and laughed and talked with the other patients. She could hear in the ordinary way about as well as the boy presented. If she was called by name she would come, even if her back were turned. The operations were performed about one year before Dr. Page published his reprint. So far as Dr. Berens could judge, he did not see what harm, outside of surgical shock, could come from the operation.

Bullet Wound of the External Auditory Canal.

Dr. Francis J. Quinlan reported a case of an Italian who had been brought to his service, at St. Vincent's Hospital, on December 24th of this year. He was a bartender by occupation, and had received a bullet wound in his head. The man was left unconscious, and an ambulance brought him to the hospital. Dr. Quinlan saw the patient the next day. The ear presented an enormous mass like a potato, and Dr. Quinlan mopped out the external parts. After two weeks of treatment the tissues were reduced in size and an exploratory operation made of the canal. The external auditory canal was entered, but still occluded the drum. The probe determined what seemed to be a hard metallic substance. The pistol had been placed as near to the external auditory canal as was possible.

After one week of waiting and constant supplication the man finally consented to an operation.

Dr. Quinlan opened the ear externally along the lines of the mastoid, and found a mass impacted in the canal up to the drum, like molten lead poured into a cavity. With the greatest difficulty in chiseling he took away a very large, irregular-shaped bullet. The man had a slight facial paralysis and considerable bleeding. The man did nicely for a week after the operation, and then complained of pain anteriorly. By this time the condition had improved and the inflammatory zone had disappeared. By careful probing upwards and forwards, Dr. Quinlan felt what seemed to be another bullet. After working for three-quarters of an hour, while he was in great pain, the doctor removed a second somewhat smaller looking bullet. The patient left the hospital one night without leave of absence. Dr. Quinlan saw him since, and the man was perfectly well with slightly impaired hearing on that side. He thought, however, that the patient had an old residual condition, which might account for the loss in hearing. Dr. Quinlan never before saw a bullet so placed as to fill up the canal and press upon it. The man was pursuing his old occupation as bartender.

DISCUSSION.

Dr. Charles E. Perkins said that he recalled a case which had come to Dr. Dench's clinic. The man had placed a revolver against and shot himself in the mastoid, and some operation had been performed to take out the bullet. They made an X-ray picture, and the condition looked as though a piece of plaster had struck the bone. There seemed to be twenty small pieces of the bullet all over that side. Some of them, Dr. Perkins said, must have been in the brain. The wound healed, the patient was fairly comfortable, there was no facial paralysis, but in looking at the X-ray one would not have thought that bone could cut a bullet into so many pieces.

Dr. Francis J. Quinlan said that in his case there were about eleven smaller pieces of the bullet impacted into the mastoid process of the bone.

Dr. John B. Rae reported the case of a bullet wound, occurring five or six years previously in a man who had shot himself with a twenty-two caliber pistol. He was unconscious and taken to a general hospital, where he stated the bullet had been removed. After leaving the hospital he was under the care of his family physician for about three weeks, and then presented himself at the Postgraduate Hospital. The patient then had a facial paralysis and an acute suppurating mastoid. There was a copious discharge of pus from the canal. Dr. Rae thought it advisable to do a radical operation, believing that the bullet had been removed. On getting into the middle ear he discovered that a large fragment, almost one-half of the bullet, had been jammed into the middle ear, just as if it had been poured in. Strange to relate, Dr. Rae said, the patient had very good hearing and a functionating vestibular apparatus. The patient reported at Christmas from San Francisco, when he stated his facial paralysis was almost gone. Dr. Rae said that the splitting up of such bullets was very interesting.

Facial Paralysis From Otitis Media Suppurativa Chronica.

Dr. Wendell C. Phillips reported a case in a young man, age thirty years, who had come to his clinic at the Postgraduate Hospital the Friday previous with a chronic suppuration of the left ear. The condition had existed for a number of years, and at the time Dr. Phillips first saw him the patient had a fairly complete facial paralysis, and stated that he had lost his hearing in the affected ear. The paralysis had occurred two and a half months previously, at which time he had a great deal of vertigo, considerable pain in his ear and nausea, which kept up for several days. The nausea and vertigo eventually subsided, there was no nystagmus, and testing the labyrinth revealed total absence of hearing and a nonfunctionating static labyrinth. There was a large mass of cholesteatoma occluding the attic and inner tympanic wall. Dr. Phillips had performed the radical mastoid operation on account of the cholesteatoma. There was no fistula of the semicircular canals. In the tympanum he found extensive necrotic areas. A large space, probably involving the section of bone between the oval and round windows, had been broken down. In touching the granulations around this area the probe had entered the vestibule. There was also much granulation tissue in the region of the promontory. No promontory was found. While curetting the granulation tissue from the floor of the tympanum a gush of blood appeared, and it was very evident that he had wounded the jugular bulb. The hemorrhage was not uncontrollable, and on packing the cavity the bleeding stopped, but later, during an attempt to complete the operation, the same return flow of blood appeared. The facial nerve exposure was found just above the oval window. He had not performed the labyrinth operation, inasmuch as nature had saved this man's life by walling off the inner territory of the labyrinth.

DISCUSSION.

Dr. John D. Richards said he could not see why Dr. Phillips had passed a probe into that labyrinth. From Dr. Phillips' remarks he gathered that there was a mass of granulation tissue covering the round and oval windows. In passing a probe under such circumstances through an oval window, it is impossible by the tactile sense to tell whether the window (granting the stapes is absent) is sealed off or not by a protecting barrier. Granting that such a labyrinth is functionally dead, it may have become so at some antecedent time, and after the original inflammatory process has subsided a normal labyrinth fluid again fills the labyrinth. We are then confronted with a functionally dead labyrinth containing a normal labyrinth fluid, which is not a surgical labyrinth. If Dr. Phillips had gone in to explore the labyrinth to find out if pus were present, he should have done more than to have merely passed a probe through the oval window.

Dr. Wendell C. Phillips said that at the time he operated the area of bone around the oval window was wide open, and that he did not push his probe into it, but the probe entered a wide open door. He said he thought that Dr. Richards was laboring under a wrong impression as to the conditions described. It had seemed to him that the whole section between the oval and round windows was gone. There was absolutely no force used. The probe dropped right in. The probe was not a small probe at all, he used it as a searcher, which just dropped into a wide opening.

Deafness Following Salvarsan Injection.

Dr. John D. Richards reported a case he had seen during the past summer, who had had syphilis some twenty odd years ago and had taken the usual three year treatment. Each year subsequently the patient had gone to Hot Springs, Arkansas, for two months. During the past summer, for some reason Dr. Richards did not know, the patient had his blood examined, and the syphilographer found it to show a positive Wassermann reaction. The patient was given salvarsan intravenously, and just about two months subsequently he awoke one morning to find that he had suddenly become completely deaf in one ear. Dr. Richards saw him on the following day. With the deafness the patient had no disturbance of equilibrium, no vertigo and no other phenomena associated with the vestibular apparatus. The affected ear was absolutely deaf and the function totally gone. Dr. Richards said that the best thing to be done was another injection of salvarsan, which the patient had injected that afternoon. Dr. Richards saw him exactly one week from the second injection of salvarsan, and the patient related the following:

Three days subsequent to his salvarsan injection, which was given on Saturday, he began to hear fairly well. On Thursday, five days after the injection, he stated he could hear as well as ever. He was an intelligent man and had tested the hearing with his watch and made the examinations himself. Dr. Richards saw him the following Saturday, just one week subsequently, when function had completely returned. He heard the forks throughout the scale, with no shortening of tone duration, and also heard the regulation whisper Dr. Richards used in his office at normal distance. There was only the shortening of a few seconds from the normal in the bone conduction. The speaker said the case was interesting from the suddenness of the onset, the nature of the lesion, and the total ablation of cochleal function without vestibular disturbance, and the quick response to salvarsan.

Dr. Richards said that the second injection of salvarsan had been given the day following the onset of the deafness. The speaker said he had seen a good many cases where the duration of deafness was over two months. He had had a case with a similar history where the deafness had lasted for six months, which had been put on several injections of salvarsan, with improvement.

DISCUSSION.

Dr. Francis J. Quinlan said he wanted to speak of a case that was given salvarsan with a luetic history, and absolute deafness had followed, the patient remaining in that condition to the present day. The man at the time of treatment received three intravenous injections, was apparently in good health and had very good hearing. According to his own citation, he lost his hearing after the second injection, and after the third was absolutely deaf.

Dr. John D. Richards said that this particular case was given salvarsan more quickly following the deafness than any other case he had ever seen, and that he never before saw a case which had recovered so quickly. Excellent results had been reported by Ellis and Swift, who drew very satisfactory conclusions in regard to the administration of salvarsan in these cases. They had relied largely upon spinal puncture; more so than on the blood examination for their tests. Dr. Richards saw a good number of these cases, where salvarsan was given some months after the deafness, in which there was a partial but very satisfactory restoration of the hearing.

Acute Mastoiditis and Ethmoid Infection.

Dr. Thomas J. Harris reported a case, in which he had been called in consultation, of acute mastoiditis, which presented several points of interest, especially on account of being affected with the prevailing epidemic of grippe. The patient was a young married woman, and the middle ear and mastoid involvement had been going on for twelve days. There was a variable temperature, reaching up to 102°, and the pulse, which was showing decided toxic elements, was also rather variable —from 120 to 130. The woman had always been well. There was also an acute ethmoid. The patient had never had any more than a certain amount of discomfort in the ear, and no mastoid tenderness at all. The suppuration was described as diminishing. The patient indeed was rather exulting in the fact that it was letting up. During the interval of the twelve days the patient had received stock vaccins and afterwards autogenous vaccins. Examination showed a general involvement of the mastoid bone. The bacteriologic findings showed a streptococcus organism. Dr. Harris said the case was interesting from the standpoint of the danger of using vaccins, and did not think a case like that warranted it. They should certainly not use stock vaccins, and he did not think that even autogenous vaccins ought to be employed, and that valuable time was being lost in depending upon them. The case illustrated that the absence of mastoid tenderness was something they could not put a great deal of emphasis on. The fact that there was a general mastoid involvement was in itself a sufficient indication for immediate interference. The case of course had been complicated by the ethmoidal condition previous to when Dr. Harris saw it. He did not know how the other gentlemen looked at it, but he thought they must take the view of operating much earlier and advising entrance into the mastoid than is usually done in such a case as this. In this case with grippe, he thought the lapse of twelve days almost unwarranted.

DISCUSSION.

Dr. Frank T. Hopkins said he would like to report a case which had come to his office with a history of a slight discharge in the ear and extreme tenderness over the mastoid, the patient not being able to sleep for two nights. The tympanic membrane was somewhat bulging. Dr. Hopkins incised it freely and gave the woman a serum injection of mixed influenza stock vaccin, and told her that if she were not better within twenty-four to forty-eight hours, a mastoid operation would have to be performed. When she came to his office the next morning the pain in the mastoid was much less. She asked that the operation be postponed another day. At the end of the second day the pain was gone and the discharge lessened. The patient went on to recovery without any further mastoid trouble. This case was suggestive in that sometimes the injections of serum were of benefit. This was not such an early case, and the trouble had been going on for one week before she was seen by Dr. Hopkins. He said that sometimes the injections of serum would work very satisfactorily.

Dr. William C. Braislin said he might refer to a case he operated on the previous Thursday. A practitioner caller him in three weeks previously to incise a membrane. A culture from this ear, the right, showed staphylococcus pyogenes aureus. The patient had received one dose of the polyvalent (Van Cott's) mixed vaccin, and subsequently got four others. The case was again seen in the fourth week of her illness, when she was operated upon immediately on account of the pronounced mastoid symptoms. They found both the mastoids in very bad shape, pus extending to the dura over the lateral sinuses. Both sinuses were exposed. This case was one of the first mastoids, Dr. Braislin said, he had seen treated with vaccins in a thoroughgoing manner for a considerable period previous to operation, and the impression gained was not favorable to its use in acute cases of mastoiditis. Cultures were also taken from both mastoids at time of operation. The right and left mastoids both contained staphylococci and streptococci, but the left more streptococci than right.

Dr. Wendel C. Phillips said that the case Dr. Hopkins reported followed exactly in the line that the majority who do not have vaccins do. Dr. Phillips would not attribute recovery to the vaccin in that case. He desired to emphasize the fact that the tendency among otologists is to delay operation on the mastoid for a longer period than they did a few years ago. Unless the symptoms were extremely severe, Dr. Phillips doubted if many men operated during the first week of the disease. He said he was greatly interested in the discussion at the San Francisco meeting of the American Medical Association, Section on Diseases of the Ear, Nose and Throat. At this meeting Dr. Norval H. Pierce made a plea for postponing all operations on the mastoid, excepting complicated ones, until the third week. He stated that there were very many operative cases that did not have very much mastoid tenderness. His experience was that in cases which had gone eight, ten or twelve days, if there is no cessation in the discharge and the temperature persisted, they had indications that pointed to the necessity for operative interference.

Dr. Thomas J. Harris said that in reporting his case he had endeavored to bring out the point that they should not let one or perhaps two symptoms determine the definite time or day of operation, or at what time they should enter the ear, whenever there are evidences of toxemia. In a case where there was a temperature and which showed general involvement and profuse discharge, any great delay was a bad thing. Often if these cases were gone into too late, various involvements would be found. Dr. Harris said he had recently seen a case where he would have been very glad if he had been called in before. There was not much temperature in this case, but a decided breaking down.

Dr. Frank T. Hopkins said that he had given vaccins in some cases within a week or ten days after the inflammation started. In one case about a year previously, in a child, the patient showed a very bad mastoid suppuration and inflammation of the other ear. The one ear had been discharging for a week with great profusion. Dr. Hopkins gave a staphylococcus vaccin, and the ear, which had been discharging for days, cleared up very quickly. The child was in excellent condition within a very short time. The speaker had given vaccins to a patient with antrum disease. The pain had been going on, not severely, but continuously, for a week or ten days, and under this treatment the antrum cleared up promptly.

Exposure of Capsule of Jaw by Cholesteatoma.

Dr. T. Passmore Berens said that while doing a Stacke operation for the students in the Manhattan Eye, Ear and Throat Hospital, he uncovered a tremendous cholesteatoma. In the first place the whole anterior wall of the canal was eroded and the capsule of the jaw was exposed. Dr. Berens did not enter it. The other interesting point was that in taking out the cholesteatoma he found the ossicles almost normally located, but absolutely bound down to the inner wall of the ear. There was no necrosis of the ossicles. There was no drum membrane, and the ossicles looked simply plastered down. She had a very stiff jaw. The wound is in a satisfactory condition.

DISCUSSION.

Dr. Charles E. Perkins said he had a case similar to Dr. Berens' the year previously. The patient gave a short history. The otologist who had referred the patient was in doubt as to whether the patient had a furuncle or not. There was a large swelling of the canal present. The patient refused to have anything done until an X-ray was taken, and if that was positive the doctor in attendance on the patient consented to an operation in the hospital. On the first report of the examination of the plate there seemed to be nothing the matter with the mastoid. Dr. Perkins was in favor of operating, but they sent her home. The pathologist looked at it the next day, after the plate had become dry, and said it was an operative case. She was brought back to the hospital, and Dr. Perkins opened the mastoid. There were two openings in the mastoid cortex in and around the ear. She could not open the jaws. She said she had first noticed something wrong with her ear about two months previously. When Dr. Perkins entered the antrum he found it filled with cholesteatomatous matter. The anterior wall, clear to the tympanic ring, was absent, and the cholesteatomatous mass buried around the condyle of the jaw. The posterior wall of the canal along the antrum was entirely broken down. There was a fistulous opening in the horizontal semicircular canal. He did not put a probe into it, but left it alone. Her hearing returned and the active symptoms disappeared and she completely recovered.

DR. T. PASSMORE BERENS said that his case was a true cholesteoma without granulation tissue.

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X.

A CONTRIBUTION TO THE BACTERIOLOGY OF THE SOCALLED COCCOBACILLUS FETIDUS OZENÆ (PEREZ), WITH ADDITIONAL NOTES ON THE TREATMENT OF CLINICAL OZENA BY MEANS OF POLYVALENT VACCINES MADE FROM THE SAME ORGANISM.*†

BY

HENRY HORN, M. D., AND ERNST ALBRECHT VICTORS, M. D.,

SAN FRANCISCO.

GENERAL INTRODUCTION.

At the sixty-sixth annual session of the American Medical Association, held in San Francisco, June, 1915, one of us (Horn) presented a preliminary report on "The Etiology and Treatment of Ozena."[148]

The work up to this time had been done at the laboratories of Stanford University and the St. Francis Hospital. Some months later, Dr. Ernst Victors, director of laboratories, San

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†Read at the twenty-second annual meeting of the American Laryngological, Rhinological and Otological Society, May 5, 1916, at White Sulphur Springs, West Virginia.

Francisco Polyclinic and Postgraduate College, was invited to join in the investigation, and the following study is the result of work done in his private laboratory. We take this opportunity of thanking Stanford University and the University of California for the generous way in which they have placed at our disposal all of the ozena material of these two institutions; and to the many physicians in various parts of the United States who have sent material or referred cases to us for observation and treatment.

We believe that the contention of Perez,[122] made thirteen years ago, that the coccobacillus fetidus ozenæ is the true and only cause of ozena, has not been successfully controverted. Our bacteriologic work has consisted in the more exact study of the morphologic characteristics of this organism; and an endeavor to correlate it with the bacillus bronchisepticus. Its assumed close relationship to the Friedlander group seems by our work to be fully settled in the negative. We believe that it has absolutely nothing to do morphologically with the bacillus of Abel or other members of the Friedlander family. That these two organisms exist side by side in the nose, there is no question. In fact, one can observe a typical Perez bacillus and the typical bacillus of Abel and Löwenberg on the same slide; and we have often noted that as the case improved clinically, the Friedlander group takes the place of the Perez bacillus and seems the last to clear up. Since the publication of our preliminary report but one article has appeared which has added any new light to the subject. The article by Burckhardt and Oppikofer[143] advances a theory which we were able to disprove many months ago—viz., that the Perez bacillus was closely related to the Friedlander group. These investigators were almost on the verge of what we consider the crux of our present paper when they casually mentioned in a footnote that one strain of the Perez bacillus furnished them by Hofer was motile. This interesting observation they credited to a contamination. The fact of the motility of this organism is now proved beyond question. Every strain of the Perez bacillus, twenty-nine cases, shows distinct motility, and flagellæ can be easily stained in all cases. The positive complement fixation reactions with strains of the bacillus bronchisepticus, isolated by Prof. Carl Meyers, University of California, puts it indisputably into the class with these organisms.

The work of Torrey and Rahe[142] satisfactorily proved the cause of distemper in dogs, and has been accepted without question by us as a basis for our investigation. It has been frequently suggested, and carefully worked out by Perez,[130] that the infection in ozena probably originates from the handling of dogs. Hofer[118] has added to his polyvalent vaccine a culture from the nose of healthy dogs, although, as far as a study of the literature goes, he does not specifically mention the bacillus bronchisepticus, nor refer to the work of Torrey and Rahe. We are now engaged in a more extensive study of this phase of the subject, but results are not ready for publication.

A review of the work of Perez, the investigators who followed him, and the more recent work of Hofer, need not be repeated here. A résumé in English by Guggenheim,[147] our own preliminary report,[148] and the article by Burckhardt and Oppikofer[143] in German, fully cover the historical phase of the subject.

It might be interesting to restate the questions which were asked in our preliminary report and see whether, in the light of our present work, the questions have been answered.

We are extremely sorry for the pessimistic and somewhat suspicious tone which pervaded our former publication, and which unfortunately was shared by others who saw the work in Vienna, for our recent work has convinced us that the solution of the etiology of ozena is almost at hand, and that very satisfactory results can be hoped for in the treatment of this disease by means of vaccine.

1. "Is the Perez bacillus the true cause of ozena?" As stated elsewhere, taking into consideration our own additions to the morphology of this organism, we consider this question has been answered in the affirmative.

2. "Has not the undoubted cure of certain cases with vaccines made from the Perez organism and the undoubted failure to cure in other cases, clinically similar, weakened our concept of the clinical entity of clinical ozena?" We believe that for the present it is convenient to divide clinical ozena into a Friedlander group and a Perez group, according to the bacteriologic findings, until such a time as we can more accurately determine what clinical relation the Friedlander group bears to the coccobacillus, or what its rôle is in the etiology of the disease. Our detailed statement of this problem in the

bacteriologic part of the paper, shows that we believe that ozena is in reality a clinical entity.

3. "Is it not possible that cases of ozena, clinically identical, may be cured by several different vaccines?" When this question was asked, we had in mind the improvement which some cases seemed to show after the administration of stock mixed vaccine containing staphylococci, streptococci, micrococcus catarrhalis, etc. Our recent work has convinced us that these saprophytes clear up as readily with the pure Perez vaccine as they did with the old mixed stock vaccines.

4. The last question as to whether we have been able to produce a successful vaccine we can answer decidedly in the affirmative. A study of our case histories would seem to substantiate this contention. We attribute our apparent failures, spoken of in the latter part of the preliminary report, to be due to the fact that we had not, up to that time, prepared a proper vaccine.

ANALYSIS OF THE CASE HISTORIES.

As will be seen by reference to Charts 1 to 5, seventy-one cases have been studied. Chart 1 shows that in fifteen cases of simple atrophic rhinitis, neither the Perez bacillus nor the bacillus of Abel was once found. This simply goes to show that these two organisms are not present in this type of case, nor are crusts and odor usually present, and also shows that the bacillus is never found in the normal nose. This last point has also been confirmed in a large series of routine laboratory examinations, the details of which are not given here, and although the Abel type is never present, other members of the Friedlander group are frequently found in accessory sinus and other infections.

This leaves forty cases (Charts 2—4) in which a diagnosis of clinical ozena was made, either by ourselves or by others. We think a great deal of reliance can be placed on these clinical diagnoses, as they were all either examined by us, sent from the University clinics, or the histories accompanying the cases were conclusive. In twenty-eight of these forty-eight cases the Perez organism was shown to be present, or fifty-eight per cent. The best previous results, given by Hofer and others, have been about thirty per cent, possibly indicating an improvement in our methods of isolation.

A most important fact seems to us to be that in the remaining forty-two per cent of the cases only the Abel bacillus or members of the Friedlander group were found. In other words, in every case where a clinical diagnosis of ozena was made, we have been able to demonstrate one or the other of these two groups as the predominating organisms.

These findings were not always obtained at the first bacteriologic examination. Often as many as fifteen examinations would be necessary before we could convince ourselves of the presence of the Perez organism. In one case (M. H., Chart 2) the bacillus pyocyaneus was found in pure culture nine times; but finally, by various methods of cultivation, we were able to demonstrate the Perez bacillus.

In fact, we have about come to the conclusion that in every case of true clinical ozena, one of these two organisms or both of them must be found, otherwise the technic is at fault. We are inclined to the belief that investigations now being made will enable us to explain why the Perez organism cannot be demonstrated in every case, and that further improvement of technic will increase the proportion of the Perez to the Friedlander group.

It will be noted that in the thirty-two cases given in Chart 2, four are given as staphylococci. In these cases the crusts were sent from distant points, such as South Dakota, Idaho, etc., and were from three to six days in the mail. The appearance of the crusts was typical of ozena, but a strong overpowering stench of sulphuretted hydrogen was present, showing that contamination had taken place, and giving the probable reason for our inability to demonstrate the Perez bacillus.

AGE AND NATIONALITY.

The ages range from nine to fifty years, showing that our cases had neither to do with the very young nor the very old. Of the thirty-two Perez cases (Chart 2) sixteen were males, fifteen were females, showing the proportion to be about equal. Fifteen of the cases were Americans, the rest being of various other nationalities, showing that in America at least the proportion of ozena among the native born is almost as large as among the foreigners. The various percentages on these points among the Friedlander ozenas (Chart 3) is approximately the same.

CASES OF SYPHILITIC OZENA.

An interesting sidelight has been thrown on the matter of syphilitic ozena (Chart 4). Through the kindness of the University of California, eight cases of active syphilis, where the clinical diagnosis of syphilitic ozena was made, were referred to me for bacteriologic study. In three of these cases the Perez bacillus was found. In the other cases, merely staphylococci. In the cases where the Perez was not found, the crusts and odor, although marked, did not in any way resemble the typical odor of the Perez organism. We consider that the odor which accompanies these cases is usually due to the destructive process of the syphilis, and not to a specific organism. These three cases only go to prove that a true ozena can coexist with active syphilis, and, as far as at present can be seen, is not favorably influenced by the most vigorous antisyphilitic treatment. The fact that the Perez bacillus was found in the congenital, tertiary, and active secondary stages shows that it is not necessarily confined to any one stage.

Whether the depraved state of the mucous membranes found in syphilis and tuberculosis predispose to infection with the Perez bacillus is a point now under study and will be reported on later.

METHODS OF EXAMINATION.

In order to get a proper perspective of the good done by the treatment, the suggestion, which has been made by previous investigators—viz., to allow the patients to continue their own treatment at home—has ordinarily been followed out. If the patients were in the habit of washing the nose once or twice a day, this was ordered continued until such a time as it was considered unnecessary by them.

Inasmuch as the majority of these patients have lost their sense of smell, the amount of odor present can be best determined by the examiner. Very little weight is given to the statement of the parents, because after about the third injection the odor disappears to such an extent that they are unable to detect it by ordinary methods.

In a marked case of ozena the odor can be distinctly perceived at from four to six feet and even more. When in the charts the expression "No odor" is used, it means that if when the examiner places his face in immediate proximity to that of the patient, after several exhalations through the nose no trace of odor can be perceived, the case is considered odor free. If, at this contact examination, a very slight but still perceptible odor is present, the case is labeled "Slight odor." In the nearly cured cases it is necessary to repeat this experiment many times, moving the face back and forth in front of the patient, in order to detect any odor, because under certain conditions the odor is not perceived for several minutes or until five or six breaths have been exhaled through the nose by the patient.

In the apparently cured cases the disagreeable odor is absolutely gone. The breath has not even the heavy smell which often accompanies chronic catarrh, but is more the character of the breath of young and healthy infants. It is necessary to note here that we imagine that we can determine a slight difference between the odor of a Friedlander ozena and a Perez ozena. The latter has a peculiar, sweetish, nauseating, lightish odor; the former, an overpowering, heavy stench. We doubt whether any scientific value can be placed on this observation, but it is interesting in the light of future work.

It seems important to distinguish between a bacteriologic and a clinical cure. As yet, except in but one or two cases, we have not been able to convince ourselves that any of the cases are bacteriologically cured. In some of the cases where the clinical improvement has been greatest, we have been unable to find the Perez bacillus. In other cases, after the Perez bacillus has disappeared, the Friedlander bacillus seems to have taken its place.

In those cases which have been several months without treatment of any kind, where the crusts and odor have entirely disappeared, where the appetite and weight have increased, and where the general statements of the patient are that they feel immensely improved and relieved, we have adopted, for the sake of convenience, the term "clinically cured." Naturally, these cases will be kept under observation for a period of at least a year before any more definite statement can be made. It must be distinctly understood that we have never claimed a case as finally cured.

Because of the expense entailed, the Wassermann examination was not demanded in every case. This subject has already been very thoroughly gone into by various authors, and we believe, with them, that syphilis plays absolutely no rôle in the etiology of ozena.

No extensive discussion will be entered into with regard to the rôle which accessory sinus disease plays in the cause of ozena. Our impression is that in practically every case the mucous membranes of the antrum and sphenoid are infected with the Perez organism. We have been able to prove in one case at least that a pure culture could be obtained from the antrum, and in many cases have been able to obtain the Perez bacillus in pure culture from the mucous membrane of the sphenoid.

That the entire mucous membrane of the nose, including the submucous tissue, and all of the accessory sinuses are involved, is further substantiated by the fact, that even in those cases where cultures from the nasal mucous membrane showed only the various saprophytic organisms, it was extremely easy to get the Perez bacillus in pure culture from the submucous tissues overlying the turbinates.

This method is as follows: The turbinate was first cocainized, then painted with pure tincture of iodin. A heavy needle, attached to a Luer syringe, was punctured through the iodin area and the needle withdrawn and a bouillon tube was inoculated. The tube always remained sterile and showed that no bacteria were carried into the submucous tissue by the needle. The needle was again punctured through the iodin area, the point moved about, near or below the periosteum of the turbinate, until a slight hemorrhage had taken place, and then the plunger partially withdrawn. The few drops of blood were placed in bouillon, and usually, in the genuine ozena cases, a pure culture of the Perez bacillus was obtained. These experiments were repeated on both the inferior and middle turbinate. We have not proved the extension to the septum and other parts of the nose, but we have every reason to believe that in a well marked case it is to be found in practically all of the mucous membranes of the nose and accessory sinuses. It would seem a far cry to attribute to the accessory sinuses a primary etiologic rôle. It seems to us far more logical to assume that the infection is an extraneous one, and spreads by contiguity from the mucous membranes of the septum and turbinates.

Although in this series of cases, because of insufficient time, meagerness of the material, and the fact that we did not wish to complicate the picture of the cure by anything other than straight vaccin therapy, the sinuses were not drained, yet we feel, that in order to produce the best results, it is absolutely essential, in all cases, to drain the sinuses whenever there is a suspicion of trouble. In fact, we believe that it would be a good plan in every case, at the beginning of the treatment, to do several punctures of the antrum with thorough irrigation. If, as is often the case, on account of the atrophy, the sphenoids can be entered, it would be advisable to thoroughly wash out these cavities. In several cases where the odor did not properly disappear, we found a retention of pus in the sphenoid or antrum, and a cleaning out of the same, would rapidly result in an odor free nose.

The striking results we have obtained by vaccine therapy in ozena is unquestioned (Chart 5), and having established this fact, we should use every possible accessory aid in the elimination of pus foci. We are convinced that no amount of vaccine treatment will ever cure a case of Perez infection in the retained pus of an antrum or a sphenoid.

METHODS OF VACCINE ADMINISTRATION.

We have found that to obtain the best results it is necessary to work out the proper interval of dose for each individual patient. To do this expeditiously, we begin every case with 200 million initial dose, and have the patient report in twenty-four hours. If no symptoms indicating reaction are reported, we give 400 million, and have the patient report again in twenty-four hours. If there are no symptoms of reaction, we give 800 million, and continue to have the patient report every twenty-four hours, doubling the dose (every twenty-four hours) until the patient reports definite symptoms of reaction (headache, general malaise, poor appetite, temperature and occasionally nausea). The dose causing this reaction we consider the minimum therapeutic dose. Usually by the time the dose is 800 million the patient reports reaction symptoms. At each injection we then have the patient report in seventy-two hours, when we give the next dose. We increase the minimum therapeutic dose by 200 or 400 million, according to the symptoms reported by the patient. The symptoms indicating non-increase of dosage or extension of the seventy-two-hour interval to ninety-six hours or more, are fatigue, depression, loss of appetite, subnormal temperature, and failure of pulse to return to rate at time of previous injection. We believe the three and four day interval will be found to give better results. Improvement should be unquestionable after the fourth dose following the dose producing reaction, and the first course of injections should number about twenty, at the above (three or four-day) interval.

In our experimental work we have used many different combinations of polyvalent Perez vaccines. It was only toward the last of our work that we were enabled by means of agglutination experiments to produce vaccines which are fairly satisfactory. In the last series we have used only vaccines whose potency has been increased by passage through animals. We have a plan in our new experiments for greatly increasing the efficiency of the stock vaccine. Hofer[130] speaks of his bitter disappointment in finding some of his results negative because of improperly prepared vaccine; and we feel that if the present series of cases could have been treated with the vaccine which we are now using, our own results would have been far more satisfactory. For these reasons some of the apparent contradictions are explained, and some of the long series of injections could have been greatly shortened.

The dosage of four to five billion seems unnecessarily large, and as the vaccine becomes more potent the number of bacteria per cubic centimeter will be greatly lessened. Even in small children we have never seen any ill effects with large doses, and we must strongly urge that in order to get the best results the dose must be pushed to the extreme limit of reaction. It is remarkable how quickly the patients, after having one severe reaction, become almost immune to subsequent doses, no matter how large.

CASE HISTORIES.

Case 1.—J. M., nine years; male; Mexican.

Family History.—Mother suffers from rhinitis atrophica fetida, Perez type. One brother same.

Past History.—Bothered since three years of age. Frontal headaches. Marked odor.

Bacteriologic Examination.—Perez.

Examination.—Marked atrophic rhinitis. Greenish crusts anterior end of middle turbinate. Odor not particularly marked.

September 22, 1914. Right side blocked with greenish crusts. Odor not marked.

October 21, 1914. Odor extremely offensive. Crusts abundant. Injection, Sherman's No. 36, 3 million.

October 28, 1914. Crusts better. Odor better. Injection, 6 million Sherman's No. 36.

December 5, 1914. Crusts decidedly better. Odor marked. Reaction slight.

December 8, 1914. Injection, 6 million No. 36.

December 19, 1914. Odor and crusts about the same as at first.

December 26, 1914. Injection, 8 million No. 36.

January 9, 1915. Injection, 7 million No. 36.

January 23, 1915. Following last injection marked local reaction. Odor much improved. Injection, 5 million No. 36.

January 30, 1915. Injection, 6 million No. 36. Some improvement.

April 10, 1915. Crusts somewhat improved. Odor still strong. Beginning stock Perez vaccine No. 1. 150 million.

April 15, 1915. Slight local reaction. No headache. 250 million.

April 17, 1915. Second day following last injection, odor markedly increased. Crusts loosened up. Odor entirely disappeared. No crusts. Injection, 300 million.

April 24, 1915. Odor still present, but not so severe as formerly. Crusts diminished. Injection, 500 million.

May 1, 1915. Considerable reaction from last injection. Crusts very much decreased. No odor. Mother marks improvement.

May 8, 1915. No crusts. No odor. Injection, 500 million.

May 15, 1915. General condition somewhat improved. Injection, 500 million.

May 22, 1915. Condition much better. Injection, 1 billion.

June 5, 1915. General condition much improved. Odor better.

June 19, 1915. No crusts. Slight odor.

March 28, 1916. Patient has been away nearly a year. It is possible to get a slight suspicion of odor if the child breathes directly in your face. There are no crusts in the nose. Mother says general condition about the same. Injection, 500 million No. 6 stock Perez. Mother says odor is decidedly improved since first treatment, but if allowed to go without washing, odor returns.

March 29, 1916. Decided local reaction. Nose not washed for twenty-four hours. Decided odor.

March 31, 1916. No crusts. No odor. Injection, 1 billion stock No. 6.

April 5, 1916. Slight reaction. No crusts. No odor. Injection, 1 billion stock No. 6.

April 8, 1916. Injection, 1½ billion. No crusts. No odor.

Note.—This was one of the early cases in which we tried stock mixed vaccines to clear up the saprophytes. The treatment at first seemed to benefit, but later the odor and crusts returned. When the Perez was started, April 15th, it was noted that after the second injection the odor became decidedly worse. This is an observation which we have made in many subsequent cases. Seven injections were given, when the case remained away nearly a year. On return the condition was markedly improved over the first examination, but the Perez was still present. The case was started with a new series of Perez vaccins.

Case 2.—H. M., aged eight years; male; Mexican; schoolboy.

Family History.—Mother suffers from rhinitis atrophica fetida. Perez type. One brother same. (J. M., Case 1.)

Past History.—Nose trouble for three years. Strong odor at all times. Crusts worse on right side.

Bacteriologic Examination.—Perez.

Examination.—Marked atrophic rhinitis. More marked on left than right. Inferior turbinate almost disappeared. Left nostril completely blocked with greenish, foul-smelling crusts. Odor very offensive. No crusts on right side.

November 21, 1914, to January 29, 1915, four injections of Sherman's No. 36.

April 13, 1915. Injection Perez No. 1, 200 million. Since last injection of No. 36, crust formation is about the same. Odor about the same.

April 17, 1915. Severe reaction from injection. Pale and nauseated. Absolutely no odor or secretion in the nose. No crusts or pus.

April 24, 1915. Injection stock, 300 million. No odor. General condition improved. No crusts.

May 1, 1915. Condition of the nose about the same. No odor. Strong reaction. Following last injection, headache for two days. Nausea and malaise.

May 8, 1915. Injection stock, 250 million. No crusts or odor in nose.

May 15, 1915. Injection stock, 250 million. No reaction. Nose the same. Hg., eighty-five per cent.

May 22, 1915. Condition about the same. Feeling of malaise following last injection.

June 5, 1915. General physical condition improved. Odor less.

June 19, 1915. No crusts or odor in nose.

March 28, 1916. Injection, stock No. 6, 300 million. Case not seen for nearly a year. Patient has been very well since last examination. No trouble with nose. Mother has not noticed odor. No odor. Left side more crusts.

March 29, 1916. Nose not washed for twenty-four hours. No crusts. No odor. Decided local reaction from last injection.

March 31, 1916. Injection stock No. 6, 1 billion. No crusts. Slight odor.

April 5, 1916. Injection stock No. 6, 1 billion. Severe general reaction from last injection. Headache, malaise and fever lasting forty-eight hours.

April 8, 1916. Injection stock No. 6, 1½ billion. Slight reaction from last injection. No crusts or odor.

Note.—The same observation which we made on J. M., his brother, applies here. H. M. always took the treatments badly. The reactions were very severe, keeping him in bed for several days at a time. The same dose on his brother, but one year older, had but very little effect. Case still under treatment.

Case 3.—B. N., aged thirteen years; female; American; schoolgirl. First examination, July 26, 1915.

Family History.—Negative. Sister complains of odor. Odor very offensive at times. Not characteristic at time of examination.

Past History.—Has had odor and crusts in nose for years.

Bacteriologic Examination.—Perez.

Examination.—Typical ozena, with crusts covering inferior turbinate, both sides. Middle turbinate, right, more atrophic than left. Marked pharyngitis. Process extends down almost to larynx. Larynx not involved. Odor marked.

July 26, 1915. Injection stock Perez, 750 million.

August 6, 1915. Injection, 1 billion. Patient says odor is better. No crusts. Scanty amount of secretion. Absolutely no odor. Slight general and local reaction.

August 16, 1915. Injection, 500 million. Crusts smaller. No odor.

August 21, 1915. Injection No. 36, 2 million, No. 38, 3 million. Examination of crusts gives Friedlander. Reaction from last Perez vaccine was marked; headache and fever. Local reaction.

August 28, 1915. Injection No. 36, 8 million. Increase in crusts. No odor or headache. Sister noticed odor yesterday.

August 30, 1915. Injection Perez, 400 million. Condition in nose much improved. No odor. Light reaction.

September 4, 1915. Injection, 400 million. Condition about the same as in last examination. Crusts much easier to blow out.

September 13, 1915. Injection, 500 million. Considerable crusts in nose; more left than right. No odor.

September 18, 1915. Injection, 500 million. Condition much improved. No odor and no reaction.

September 25, 1915. Injection, 1 billion. Slight headache following injection. Feels about the same generally. Absolutely no odor. Monday following last injection, noticed a decided odor.

October 2, 1915. Injection, 1 billion. Very little odor. Crusts very slight and easy to remove. Right sphenoid opened.

October 9, 1915. Injection, 1 billion.

October 18, 1915. Right antrum washed; probe puncture. Pus very thick; almost impossible to wash out.

Note.—This case received about twelve injections of the earlier vaccines. The child was very stubborn, hard to handle, and the father suspicious of the vaccine treatment. The parents noted the improvement, which, at the last examination, gave slight odor; crusts slight and easy to remove. The patient objected to the treatment to the right antrum, and did not return for further injections.

Case 4.—J. P., aged twenty years; female; American.

Family History.—Negative.

Past History.—When ten years of age, noticed odor, with great amount of crusts. Crusts very hard to remove. Gradually growing worse. Various types of treatment, with apparently no benefit. No operations. Wassermann negative.

Bacteriologic Examination.—Pure Perez.

Examination.—Marked high grade atrophic rhinitis. Typical ozena of nasopharynx. Saddle nose. Nauseating odor.

Treatment—September 8, 1915. Injection, 1 billion stock Perez.

October 9, 1915. Reaction general; chills, headache and fever.

October 15, 1915. Injection, 1 billion. Patient has not douched nose for one day. Very few crusts. No odor.

October 18, 1915. Injection, 2 billion. Increase of odor. No crusts.

October 25, 1915. Injection, 2 billion. No odor or crusts.

November 3, 1915. Injection, 2 billion. No odor or crusts. Gained five pounds.

November 12, 1915. Injection, 250 million autogenous.

November 18, 1915. Injection, 1 billion autogenous. Nose not washed for forty-eight hours. No odor or crusts.

December 1, 1915. Injection, 1 billion autogenous. Gained three pounds. No crusts or odor.

December 9, 1915. Injection, 1 billion autogenous. Nose not washed for one day. Decided odor, but no crusts. Patient says that she has had more or less odor.

December 13, 1915. Injection stock No. 3, 1 billion. Strong odor, as she had at beginning of treatment. Very few crusts.

December 21, 1915. Injection stock vaccine No. 3, 1 billion. Odor remains slight but marked.

December 28, 1915. Injection stock No. 3, 1 billion. No odor or crusts.

January 4, 1916. Injection stock No. 3, 1 billion. Nose washed daily. Very slight odor and few crusts.

January 10, 1916. Injection autogenous No. 2, 500 million. Slight odor and crusts.

January 14, 1916. Injection autogenous No. 2, 1 billion. No crusts or odor.

January 18, 1916. Injection autogenous No. 2, 1 billion. No odor or crusts.

January 25, 1916. Injection autogenous No. 2, 1 billion. No odor or crusts.

February 1, 1916. Injection autogenous No. 2, 1 billion. No odor or crusts.

February 4, 1916. Injection autogenous No. 2, 1 billion. No odor or crusts. Has not washed nose for two days. Does not have headaches any more. Patient in every way greatly improved.

Note.—This is the most brilliant result obtained in our series. When first seen, the girl was almost septic; the bridge had sunken, she was afflicted with a profound melancholia, and had very little appetite. The odor was terrible, and one was unable to stay in the same room with her. She had received various forms of treatment ever since she was a child. When examined, April 15th, or two months after last injection, she had gained nineteen pounds, her complexion was rosy, her disposition was happy, and her appetite had returned. There has been absolutely no odor or crusting during this time, and she considers herself entirely well. No further treatments will be given until some of the old symptoms are complained of.

Case 5.—W. S., aged twenty-eight years; male; Russian Jew; machinist.

Family History—Unknown.

Past History.—Has complained for years of stinking catarrh. Does not know how long. History of playing with dogs unknown. Notices himself that the odor is gradually growing worse. Says it is especially bad in wet weather.

Bacteriologic Examination.—Perez.

Examination.—Typical ozena fetida. Odor at four feet. Extreme atrophy. Few scattered whitish crusts over the whole of nose. Wassermann negative.

Treatment.—March 9, 1916. Injection stock Perez No. 6, 400 million.

March 10, 1916. General reaction. Odor decidedly worse. Loss of appetite, headache and tired feeling. No injection.

March 11, 1916. Injection stock No. 6, 600 million. Odor still present, but decidedly less. Crusts much improved. Small crusts on middle turbinate.

March 13, 1916. Injection No. 6 stock, 1 billion. Few crusts. Odor barely perceptible.

March 15, 1916. Injection No. 6 stock, 2 billion. Examination of cultures show Perez positive. No crusts or odor. Slight amount of pus in region of ethmoids.

March 17, 1916. Injection No. 6 stock, 3 billion. Odor strong. Nose perfectly clean, except for one crust on the anterior end of middle turbinate. No reaction.

March 20, 1916. Severe general reaction from last injection, which lasted up to today. Loss of appetite, headache and fever. No local reaction.

March 28, 1916. Injection No. 6 stock, 4 billion. Absolutely no crusts. Slight suspicion of odor. Patient for the first time noticed his own odor.

March 29, 1916. No crusts, slight odor; no reaction.

March 31, 1916. Injection stock No. 6, 3 billion. Absolutely no odor or crusts. Mucous membranes bright red in color and general feeling greatly improved.

April 5, 1916. Injection No. 6 stock, 4 billion. Condition same.

April 8, 1916. Injection stock No. 6, 5 billion. No reaction. No crusts, but slight odor.

Note.—This is one of the most interesting cases under treatment. The reactions were always marked, the odor was worse after the second injection and entirely cleared up after the third. In this case, for the first time we have been able to convince ourselves that the mucous membranes of the nose change from a decided atrophic condition to normal looking. Several careful observers who saw this case April 14, 1916, were unable to diagnose an atrophic condition, but considered the mucous membranes almost normal. One of the striking features of this case is the tremendous increase in the general wellbeing of the patient.

Case 6.—J. S., aged thirty-five years; male; American.

Family History.—Negative.

Past History.—Gives no history of trouble in nose. Decided huskiness of voice for last twelve years.

Bacteriologic Examination.—Perez.

Examination.—Larynx has small node on the right cord. The interarytenoid space distinctly thickened, always covered with slimy secretion and crusts. It is very difficult to make out whether or not there is an active ulceration. Both cords red and inflamed, and thickened and covered with a shiny crust. Marked atrophic rhinitis nonfetida. Very little crust over middle turbinate; not characteristic of ozena. Wassermann negative.

Treatment.—February 18, 1916. Injection stock No. 5, 100 million.

February 19, 1916. Injection stock No. 5, 100 million. No reaction.

February 21, 1916. Injection stock No. 5, 1 billion. For the first time since treatment, crust has disappeared from the anterior commissure of the larynx. Mucous membrane of the larynx more moist and healthy looking.

February 23, 1916. Injection stock No. 5, 1 billion. No reaction. Voice and larynx decidedly cleared. No crusts in interarytenoid space. No crusts in nose. Last examination of culture shows only staphylococcus.

February 25, 1916. General reaction, with malaise. Local reaction. No crusts in interarytenoid space. Larynx mucous membrane decidedly more pinkish; all signs of congestion lost.

February 26, 1916. Injection stock No. 5, 1 billion.

Note.—This case was certainly not one of clinical ozena. The stubbornness with which the larynx condition responded to treatment led us to examine the nasal secretion, where the Perez was found in pure culture. The five injections of Perez vaccine had a marked effect on clearing up the laryngeal condition. From an examination of the larynx, we would not have suspected a laryngeal ozena, and examination from the secretion of the larynx never showed the Perez organism.

Case 7.—S. P., aged twenty-seven years; female; German.

Family History.—Negative.

Past History.—When fifteen years of age first noticed disagreeable odor in nose. When twenty years old noticed headaches, from which she has suffered ever since.

April, 1910, curetted anterior ethmoids on right side. Following this, in June and October, intranasal operations on right side. February 4th, double Killian operation. Stinking pus from right ethmoid, frontal and sphenoid. Three weeks after operation left sphenoid opened.

Bacteriologic Examination.—August 15, 1915. Pure Perez.

April 6, 1912. Left sphenoid and ethmoid cells curetted as widely as possible.

April 26, 1912. Autogenous vaccine made from sphenoid shows Friedlander in pure culture. Injection autogenous vaccine, 10 million. From August 26, 1912, to October 1, 1912, twenty injections of autogenous vaccines, with varying success. Stopped treatment for two years. On return, condition same. Started another course of ten injections of autogenous vaccines without benefit, finshing March 23, 1915.

April 29, 1912. Injection autogenous, 20 million.

April 5, 1915. Injection mixed Perez, 500 million. No crusts, no odor.

April 19, 1915. Injection stock, 2½ billion. Nose much improved.

April 26, 1915. Injection stock, 4 billion. No reaction.

May 6, 1915. Injection, 4 billion stock. No reaction.

May 12, 1915. Injection stock, 4 billion. No reaction.

May 18, 1915. Injection stock, 4 billion. No reaction.

May 25, 1915. Injection stock, 4 billion. No reaction. Patient says she feels better now than she has for years.

July 17, 1915. Injection stock, 750 million.

July 24, 1915. Injection stock, 1 billion. Very slight reaction.

August 14, 1915. Injection stock, 250 million and one-half cubic centimeter of No. 36.

August 18, 1915. Injection stock, 1 billion. No reaction.

August 25, 1915. Injection stock, 1 billion. No reaction.

September 1, 1915. Injection stock, 1 billion. No reaction.

September 15, 1915. Injection stock, 1 billion. No reaction.

September 22, 1915. Injection stock, 1 billion. No reaction.

September 29, 1915. Injection stock, 1 billion. No reaction. No crusts or odor. Washes nose every day.

October 6, 1915. Injection stock, 1 billion. No reaction.

October 15, 1915. Injection stock, 1 billion. No reaction. Patient douches nose every other day.

October 20, 1915. Injection stock, 2 billion. No crusts in nose. No odor.

November 10, 1915. Injection stock, 2 billion. Patient had a severe reaction following last injection.

Note.—This case has been under treatment since April, 1912. She had an extensive double pansinuitis. Innumerable intranasal operations, including double Killian, were done for the relief of the discharge and headaches. It was not until August, 1915, that we suspected a Perez infection. Up to that time she had had many autogenous vaccines given, apparently with little benefit. The case cleared up wonderfully with the Perez vaccine, and has not been seen for several months. The physical improvement in this patient is perfectly remarkable, but all the credit cannot be given to the Perez vaccin. It is a type where the combined surgical and vaccine treatment gave the best results.

Case 8.—C. W., aged twenty-nine years; male; American; dentist.

Family History.—Negative.

Past History.—Has had typical ozena odor since nineteen years of age. No improvement, in spite of various vaccine treatments. Condition about the same for last few years. Denies lues. Strong sweetish odor on breath. Loss of smell.

Bacteriologic Examination.—Perez.

Examination.—Marked atrophy of both sides. Whitish crust over opening of sphenoids. Process more extensive on left side. Marked sweetish odor.

Treatment.—December 24, 1915. Injection No. 3 stock Perez, 250 million.

January 20, 1915. Injection autogenous Perez, 500 million. No reaction.

January 28, 1916. Injection autogenous, 500 million. No reaction.

February 4, 1916. Injection autogenous, 1 billion. No reaction.

February 12, 1916. Injection autogenous, 1 billion. No reaction.

February 19, 1916. Injection autogenous, 1 billion. No reaction.

February 27, 1916. Injection autogenous, 1 billion. No reaction.

February 29, 1916. Patient says he noticed a decided improvement in crusts. Has gained three pounds. Friends have not noticed odor. Examination: Right side perfectly dry and clean. Mucous membranes fairly moist. Left side no sign of crusts. Small amount of mucopus in back of throat. Absolutely no odor, even when patient breathes directly into face. Probably some involvement of the left sphenoid.

March 3, 1916. Bacteriologic examination of secretion shows Perez bacillus no longer present.

March 4, 1916. Injections of autogenous vaccine as follows: March 4, 1½ billion. March 8, 1916, 2 billion; marked reaction; slight local, but a general tired and malaise feeling. March 11, 1916, 2 billion; more reaction than with the last injection.

March 13, 1916. Patient previous to treatment with vaccines had both frontals, sphenoids, ethmoids, and left antrum opened intranasally. Nose perfectly clean, with the exception of a small crust in the region of the left sphenoid. Culture taken from left sphenoid. Very slight, but barely perceptible, odor when directly in contact with the face, and then only at times. There is undoubtedly more or less infection in left sphenoid. General condition very much improved, appetite good, weight about the same. Patient is able to work on his patients with every degree of comfort. No one has mentioned anything about odor. Left sphenoid reopened. No sign of pus in same.

March 28, 1916. Injection No. 6 stock, 2½ billion. Slight secretion in region of left sphenoid. Absolutely no crusts or odor. Dr. Waiss present at this examination, and also detects no odor.

March 15, 18 and 22, 1916. Injections of No. 6 stock, 2 billion.

Note.—This is probably one of the two (vide, J. P.) most brilliant clinical cures in our series. The patient, a dentist, had been obliged to give up his professional work, and had been treated by various forms of vaccine therapy, but without any benefit. At the last examination he had gained six pounds. There is absolutely no trace of odor on his breath, his general condition is markedly better, and he has received no treatment of any kind for over two months. He considers himself cured. Treatment will be omitted until some of the old symptoms develop.

Case 9.—A. J., aged thirty-four years; male; Russian; tailor.

Family History.—Negative.

Past History.—Since twelve years of age, foul smelling ozena, gradually getting worse. Several intranasal operations without benefit. Sprays nose every night. Weight, one hundred and forty-four pounds. Odor strong but not characteristic of Perez; possibly Friedlander. Has lost sense of smell. Wassermann negative.

Bacteriologic Examination.—Staphylococci.

Examination.—Inferior turbinate and middle turbinate hard to make out. Mucous membrane very thin and covered with greenish crusts. Odor perceptible at three feet.

Treatment.—February 16, 1916. Injection No. 3 Perez stock, 100 million.

February 17, 1916. Injection No. 3 stock, 400 million. No reaction.

February 18, 1916. Injection No. 3 stock, 500 million. No reaction.

February 19, 1916. Injection No. 3 stock, 600 million. Slight reaction from last injection. If patient breathes directly into face, one gets odor.

February 21, 1916. Injection No. 5 stock, 500 million. Slight suspicion of odor when in direct contact with nose. Crusts practically disappeared, except in region of the sphenoid.

February 23, 1916. Injection stock No. 5, 400 million. Odor entirely disappeared. One small crust on the anterior end of the middle turbinate. No reaction.

February 25, 1916. Injection No. 5 stock, 500 million. No reaction from last injection. No crusts or odor.

Note.—This was a typical clinical ozena, but the Perez or the Abel bacillus was never demonstrated. Seven injections gave marked improvement, but the case did not return for treatment.

Case 10.—A. M., aged twenty-one years; female; housewife.

Family History.—Negative.

Past History.—Stinking catarrh for last eight years. Played with dogs as a child. Not improved with treatment. Washes nose once a day and sprays nose once a day.

Bacteriologic Examination.—Perez.

Examination.—Extremely extensive atrophic rhinitis, with whitish crust on the anterior turbinate, and foul smelling pus. Odor perceptible at three feet.

Treatment.—March 9, 1916. Injection stock vaccine No. 6, 200 million.

March 10, 1916. Patient declares herself decidedly improved after first injection. Head feels clear, nose not so stopped. Crusts about the same. Odor is decidedly improved. Injection, 400 million stock No. 6.

March 11, 1916. Injection stock No. 6, 600 million. Shows slight general reaction. No odor or crusts.

March 13, 1916. Absolutely no odor. A few whitish crusts on anterior end of middle turbinate. Had a severe headache for a couple of hours on March 11th, from injection. Injection stock No. 6, 1 billion.

Note.—After four injections the improvement was simply wonderful, but unfortunately the case did not return for further treatment. Patient expressed herself as cured.

Case 11.—B. A., aged twenty years; male; American.

Family History.—Negative.

Past History.—Since fourteen years of age has complained of crusts and odor. People around him say that the odor is very disagreeable. Patient says he had very heavy crusts. The patient has had a regular obsession regarding his odor. In his profession as priest he found it necessary to douche his nose six to eight times a day, using several quarts of water at a time, and then was unable to satisfactorily get rid of the crusts.

Bacteriologic Examination.—December 1, 1915, Perez. January 6, 1916, Perez. February 19, 1916, Friedlander and staphylococcus aureus. February 22, 1916, pure Friedlander.

February 23, 1916. No odor, few crusts on anterior end of middle turbinate. Patient is now taking another course of stock vaccine No. 5.

Treatment.—Has had about twelve to fifteen injections. No injections since December 15, 1915. Has not washed nose for forty-eight hours. Absolutely no odor. Small whitish crusts.

Note.—The present condition of the patient is a marvelous change from his former unhappy condition. He is able to conduct his professional work without the fear of disgusting the people he comes in contact with, and as a matter of habit douches his nose but once a day. Since last December the treatment has been irregular, and he still receives, mainly for experimental purposes, an injection about every fourteen days.

Case 12.—J. A., aged twenty-two years; male; Russian Jew. Referred by Dr. G. Richards, Fall River, Mass.

Family History.—Mother has ozena.

Past History.—For four years atrophic rhinitis, with odor. Odor very offensive. Crusts very thick and hard to blow out. Douches nose every day.

Bacteriologic Examination.—November 29, 1915, Perez.

Examination.—High grade atrophic rhinitis. No crusts or odor.

Treatment.—November 29, 1915. Injection stock Perez, 500 million. Nose not washed for three days. No typical ozena crusts. Strong offensive odor.

December 3, 1915. Injection stock, 1 billion. Nose not washed for four days. Characteristic odor, few crusts. Patient has not noticed odor since last treatment. No reaction.

December 14, 1915. Injection stock No. 3, 1 billion. No crusts or odor.

December 24, 1915. Injection stock No. 3, 1 billion.

December 30, 1915. Perez positive.

January 3, 1916. Injection stock No. 3, 1 billion. Nose not washed for twenty-four hours. No crusts or odor.

January 10, 1916. Injection stock No. 3, 1 billion.

January 13, 1916. Perez positive.

January 17, 1916. Injection stock No. 3, 1 billion. No reaction.

January 24, 1916. Injection stock No. 3, 1 billion. No crusts or odor. Gaining in weight and appetite. Feels decidedly better.

February 1, 1916. Injection stock No. 3, 1 billion. No reaction.

February 8, 1916. Injection stock No. 3, 1 billion. Nose not washed for nine days. No odor, very slight crusts.

February 19, 1916. Absolutely no odor or crusts. Nose washed once in two weeks, although not necessary.

March 4, 1916. No crusts or odor. Nose not washed for two weeks.

March 13, 1916. Perez positive.

April 3, 1916. No injection since February 8th. No crusts or odor.

Note.—This case is a good example of a clinical but not bacteriologic cure. He is a Perez carrier, and although he has had no injections since February 8th, and no odor has been noted since December 14th, we can at any time demonstrate Perez in pure culture.

Case 13.—H. F., aged thirty-three years; male; French: porter.

Family History.—Unknown.

Past History.—Atrophic rhinitis. Patient complains that crusts and odor are worse during wet weather. Used to complain of headaches, but since he has been washing nose does not notice them. Washes nose with douche once a day.

Bacteriologic Examination.—Perez.

Examination.—October 29, 1915. Atrophic rhinitis both sides. Cultures taken from nose show Perez. When examined had no crusts or odor. Told not to wash nose and report in one month.

November 4, 1915. Injection No. 1 Perez stock, 500 million. Examination: Marked atrophic rhinitis. Few scattered crusts of whitish color on anterior end of middle turbinate and in region of sphenoid. Medium but characteristic odor. Patient has lost entire sense of smell. Patient's friends notice odor.

November 6, 1915. Slight local reaction. Nose same.

November 13, 1915. Injection Sherman's No. 36, 0.6 cubic centimeters.

November 16, 1915. Autogenous vaccine, 250 million. Crusts slight, odor strong. Has not washed nose for three days.

March 11, 1916. Patient returns, saying that condition is the same as before, and is obliged to wash his nose every day, and crusts and odor are as bad as ever. Examination: Condition about the same as in last examination; it is impossible to get but a small crust for examination. The nose is moist, and although patient washed nose this morning, there are no crusts or odor.

March 11, 1916. Injection No. 6 Perez stock, 400 million.

March 13, 1916. Injection No. 6 stock, 800 million. Slight local reaction. Condition about the same.

March 14, 1916. Injection No. 6 stock, 1 billion. Slight local reaction.

March 15, 1916. Injection, No. 6 stock, 2 billion. No crusts or odor.

March 18, 1916. Injection No. 6 stock, 2½ billion. Feels decidedly better, has not washed nose for three days. No crusts or odor. No reaction.

March 20, 1916. Injection No. 6 stock, 3 billion. Patient says he is feeling decidedly improved. No crusts or odor. No reaction.

Note.—This case has been treated for years in various clinics. The odor and crusts were slight at all times, and although he showed a pure Perez and admitted improvement under treatment, he fails to return for his injections.

Case 14.—M. G., aged twenty-one years; female.

Family History.—Negative.

Past History.—Never played with dogs. Parents noticed odor and crusts from age of six years. Gradually grew worse. Several intranasal operations; no benefit. Sherman's No. 36, long course, very little benefit. June, 1915, to August 26, 1915, ten injections of Perez mixed stock, with marked improvement of all symptoms and odor. Headaches have entirely disappeared and have not returned. People around her noticed same improvement. Before taking vaccine it was necessary to go and have crusts removed twice a week, and odor was unbearable. For last four weeks crust have not been removed from nose. Crusts are now so loose that she can douche them out. This could not be done before injections. Douches nose t. i. d.

Examination.—Tremendous improvement so far as crust is concerned. Typical atrophy. Slight whitish crust middle turbinate and septum. Slight odor.

Treatment.—August 26, 1915. Stock Perez, 500 million.

September 2, 1915. No odor. Pus from sphenoid both sides. Injection of 500 million.

September 11, 1915. Condition same. Injection of 500 million.

September 18, 1915. Slight reaction. 500 million injection. Slight crust. No odor.

September 27, 1915. Injection of 1 billion.

September 28, 1915. Right sphenoid opened.

October 4, 1915. Injection of 1 billion.

October 11, 1915. Injection of 1 billion.

October 19, 1915. Injection of 2 billion. Crusts in region of sphenoid.

October 26, 1915. Injection of 2 billion. No odor; slight crust.

October 27, 1915. Severe reaction from injection.

November 3, 1915. Injection of 2 billion.

November 13, 1915. Injection of 2 billion.

November 24, 1915. Injection of autogenous, 250 million. Examination: Crusts in region of sphenoid. Patient unable to wash out crust. Slight odor.

December 3, 1915. Injection of autogenous, 500 million.

December 13, 1915. Sterile antrum puncture gives pure Perez. Nasal secretion pure Perez.

December 21, 1915. No crusts or odor. Appetite good. Gained four pounds. Secretion in region of sphenoid. Injection of autogenous, 1 billion.

December 27, 1915. No odor or crust. Crust easier to remove. Injection of No. 3 stock, 1 billion.

Note.—The patient received in all fifteen injections of various Perez vaccines. The condition at the beginning of the treatment was extremely unsatisfactory, the patient having had a great deal of operative work in the nose and was extremely hysteric. Her blood coagulation point was low, so that twice following work on the sphenoid the nose bled for nearly a week. When last seen there was no odor, no crusts, weight and appetite greatly improved, and general nervous condition better. The patient asked to be allowed to omit treatment for a period of six months. In this case we were able to show both in the antrum and sphenoid a pure culture of the Perez.

Case 15.—M. H., aged twenty-one years; female; American; stenographer.

Family History.—Negative.

Past History.—Odor and crusts since childhood. In 1910 crusts became much worse. Played and kissed dogs as a child.

Bacteriologic Examination.—Pyocyaneus. Six examinations from July 15, 1915, to December 4, 1915. December 4, 1915, Perez positive.

Examination.—Marked atrophic rhinitis. No crusts. Slight amount of pus.

Treatment.—August 13, 1915. Injection stock No. 1, 500 million.

August 21, 1915. Injection No. 36, 3 million; No. 38, 3 million.

August 24, 1915. Injection No. 36, 5 million; No. 38, 5 million.

August 28, 1915. Injection No. 38, 8 million; No. 36, 8 million. Condition very much improved. No crusts or odor.

August 30, 1915. Injection No. 2 stock, 600 million.

September 3, 1915. Slight reaction. Headache, fever. Slight local reaction. Crusts easier to blow out. No odor.

September 6, 1915. Injection No. 2 stock, 600 million. Considerable reaction from last injection.

September 18, 1915. Injection No. 2 stock, 1 billion. Condition much improved.

September 25, 1915. Injection No. 2 stock, 1 billion. Patient has feeling of lightness and well-being. Mucous membrane very dry.

October 2, 1915. Condition about the same. Injection of 1 billion.

October 4, 1915. No reaction. Considerable crusts on right side.

October 9, 1915. Injection of 1 billion. No crusts, no odor and no headaches.

October 16, 1915. Injection of 1 billion.

October 23, 1915. Injection of 2 billion No. 2 stock. Nose in very good shape.

October 30, 1915. Injection of 2 billion. No odor and no crusts.

November 6, 1915. Injection of 2 billion. Very little crusts. No odor.

November 13, 1915. No odor, no crusts and no headaches. Has not washed nose for one week. Injection of 2 billion.

November 20, 1915. No crusts. No odor. Instructed to not wash nose for one week.

November 29, 1915. Blood, crusts and submucous cultures taken. No crusts or odor. Last examination by Dr. Victors and myself shows still no Perez organism.

December 6, 1915. Injection of 1 billion stock No. 3. No crusts or odor.

December 13, 1915. Patient says she had considerable odor all week. Douched the nose today. No crusts and no odor. Injection of 1 billion No. 3 stock.

December 20, 1915. Slight reaction. Slight dizziness, nausea and headache. No odor. Injection stock No. 3, 1 billion.

January 5, 1916. Patient has not washed nose since last visit. No odor, very few crusts. Patient is losing weight and appetite. Injection stock No. 3, 1 billion.

January 19, 1916. Injection No. 3 stock, 1 billion. Patient feels extremely well two days following injection.

January 27, 1916. Condition same. Injection stock No. 3, 1 billion.

February 4, 1916. No odor or crusts. Patient discharged for one month.

Note.—In this case nine examinations were made before the Perez could be demonstrated. There was always a very abundant growth of bacillus pyocyaneus. Stock vaccines were tried, to clear up the pyocyaneus infection, but without benefit, and it was not until a Perez vaccine was used that the case improved until she was discharged clinically cured. In all, eighteen injections were given. In this case the submucous examination showed the pyocyaneus.

Case 16.—H. K., aged twenty-five years; female; Irish.

Family History.—Negative.

Past History.—Had odor and discharge since childhood. Odor has not improved, but seems to be getting worse. Did not play with dogs. Odor extremely offensive, crusts entirely blocking nose.

Bacteriologic Examination.—July, 1915, Perez. September 22, 1915, Perez. February 14, 1916, Friedlander and Perez on same plate.

Treatment.—From July to November about twenty injections by Dr. Victors. When the case was examined by me she was free from odor and crusts, and was generally very much improved and satisfied. The case is still receiving injections from time to time for experimental purposes.

Case 17.—A. L., aged thirty-five years; male; German. First eamination, April 8, 1916.

Family History.—Negative.

Past History.—The day case appeared for examination no crusts or odor. No diagnosis of ozena could be made other than marked atrophic rhinitis.

Bacteriologic Examination.—Perez.

Examination.—Marked atrophic rhinitis.

Treatment.—April 8, 1916. Injection stock No. 6, 500 million.

April 10, 1916. Injection stock No. 6, 1 billion. Slight local reaction.

Note.—April 5, 1916. When seen today, reports fairly satisfied. Feels generally much improved, has no odor or crusts. Case still under treatment.

Case 18.—C. M., aged thirty-nine years; female; Mexican; housewife.

Family History.—Negative.

Past History.—Dry catarrh. Crusts always right side. Formerly strong odor, but none at present.

Bacteriologic Examination.—Perez.

Examination.—Marked atrophic rhinitis. Inferior turbinate almost disappeared; also the middle turbinate. Covered with dark greenish crusts. Odor slight but characteristic.

Treatment.—March 31, 1916. Injection stock No. 6, 500 million.

April 5, 1916. Injection stock No. 6, 1 billion. Local reaction. No odor or crusts.

April 8, 1916. Injection stock No. 6, 1½ billion. Slight reaction. Condition about the same.

Note.—This woman brought her two sons to the clinic. (Cases 1 and 2.) An examination of the mother, as above given, gave a pure Perez. The woman did not complain of any nasal trouble. Case still under treatment.

Case 19.—R. P., aged seven years; female; Italian; schoolgirl.

Family History.—Negative.

Past History.—Strong foul odor all her life.

Bacteriologic Examination.—Friedlander.

Examination.—Marked atrophic rhinitis. The entire nose is filled with crusts. Crusts are blackish, very foul smelling. Odor is perceptable at four to six feet.

Treatment.—March 24, 1916. Injection stock Perez No. 6, 500 million.

March 25, 1916. Injection No. 6 stock, 800 million. No reaction.

March 27, 1916. Injection No. 6 stock, 1 billion. Odor and crusts decidedly better. No reaction.

March 30, 1916. Injection stock No. 6, 1 billion. Neighbors say odor is better.

March 31, 1916. Injection stock No. 6, 1½ billion. No reaction.

April 4, 1916. Injection stock No. 6, 1½ billion. Slight odor and crusts.

April 8, 1916. Injection No. 6 stock, 2 billion. No reaction. Condition same.

Note.—This case shows the large Friedlander bacillus, and, like, the V. M. case, is extremely unsatisfactory in treatment, although marked improvement is noted.

Case 20.—L. P., aged twenty-eight years; male; Italian; fisherman.

Family History.—Negative.

Past History.—From age of six years complained of small crusts and odor, with headaches.

Bacteriologic Examination.—Friedlander.

Examination.—Medium atrophic rhinitis. Absolutely no crusts. Slight but characteristic odor of ozena. Nose not typical of ozena. Never washes nose. Typical large black crusts in the nasopharynx, which are very difficult to remove.

Treatment.—February 19, 1916. Condition unchanged. Odor scarcely perceptible. Marked glazed look to the nasopharynx. Larynx dry but not typical of ozena. Injection stock Perez No. 5, 500 million.

February 21, 1916. Injection No. 5 stock, 500 million. No reaction.

February 26, 1916. Injection No. 5 stock, 500 million. No reaction.

February 29, 1916. Injection No. 5 stock, 800 million. Crusts about the same. No odor. Slight local reaction.

March 1, 1916. Injection No. 5 stock, 1 billion. Crusts and odor about the same.

March 4, 1916. Injection No. 5 stock, 2 billion. Slight local reaction.

March 6, 1916. No crusts or odor. Nasopharynx same. General reaction without headache. Patient complains of weakness in knees and loss of appetite. No injection.

March 9, 1916. Injection No. 6 stock, 2 billion. No crusts or odor.

March 14, 1916. Injection No. 6 stock, 2 billion. Nasopharynx beginning to show a slight degree of moisture, otherwise condition the same.

Note.—This case has not reported for treatment for over a month. At last examination there seemed to be a slight improvement in the pharyngeal condition. The odor, which was always very slight, did not return.

Case 21.—A. D., aged thirty years; female; Italian.

Family History.—Negative.

Past History.—Has complained of this disease since childhood; gradually grown worse. Did not play with dogs as a child. Patient has retained smell. Smells her own odor. Odor characteristic of Friedlander.

Bacteriologic Examination.—Friedlander.

Examination.—Marked atrophic rhinitis. Crusts on left middle turbinate. None right.

Treatment.—February 24, 1916. Injection Perez stock No. 5, 100 million.

February 25, 1916. Injection No. 5 stock, 200 million. Crusts about the same. Odor possibly slightly improved. Patient says she feels better. Had slight local reaction.

February 28, 1916. Injection No. 5 stock, 500 million. Slight local reaction.

March 1, 1916. Injection No. 5 stock, 1½ billion. Crusts much drier. No free pus. Very slight odor noticed only by approaching near to nose.

March 3, 1916. Injection No. 5 stock, 2 billion. Slight local reaction. No headache. Crusts decidedly improved. Slight but faint odor.

March 6, 1916. Distinct general reaction from last injection. Pain in arm, general malaise and loss of appetite. No injection.

March 9, 1916. Injection No. 6 stock, 2 billion. Very few crusts. Very slight but typical odor.

March 13, 1916. No injection. Odor practically gone. On account of patient's general reaction, injections stopped for nine days.

March 24, 1916. Injection No. 6 stock, 2 billion. One large crust on left side without odor; right side entirely clean. Slight odor.

March 30, 1916. Injection No. 6 stock, 2 billion. Slight odor. Few crusts.

April 3, 1916. Injection No. 6 stock, 3 billion. Slight odor, few crusts.

April 10, 1916. Injection No. 6 stock, 3½ billion. No crusts, slight odor.

Note.—This is what we call the septic type. The color is bad. She takes her injections badly, and, as is often the case with this form of infection, the result is slow and the improvement not nearly so satisfactory as in the Perez infections.

Case 22.—E. D., aged thirteen years; male; American; schoolboy.

Family History.—Unknown.

Past History.—The odor first noticed on breath three years ago, and has gradually grown worse. Has large amount of crusts in the nose, which are very difficult to remove. Odor very offensive. For the last six months has been decidedly worse.

Bacteriologic Examination.—Friedlander.

Examination.—Hypertrophy of both sides. Nose entirely filled with large crusts. Odor is extremely offensive; perceptible at five feet. The odor is that of a Friedlander more than a Perez.

Treatment.—March 28, 1916. Injection stock Perez No. 6, 500 million.

March 29, 1916. Injection No. 6 stock, 1 billion. Decided local reaction.

March 31, 1916. Injection No. 6 stock, 1 billion. Strong odor. Nose entirely blocked with crusts.

Note.—Case still under treatment.

Case 23.—M. Z., aged thirty-two years; female; Hungarian.

Family History.—Negative.

Past History.—At eighteen years of age noticed odor and crusts. Sense of smell and taste lost.

Bacteriologic Examination.—Perez.

Examination.—Saddle nose. High grade atrophic rhinitis. Impossible to make out turbinates. Entire nose covered with dark crusts. Odor extremely offensive.

Treatment.—March 12, 1915. Injection stock, 100 million.

April 7, 1915. Injection stock, 250 million.

April 14, 1915. Injection stock, 250 million. Odor decidedly better; crusts about the same.

Note.—This case, an ignorant Hungarian woman, expressed herself as believing that vaccine treatment was a form of poison and refused further injections.

Case 24.—E. O., aged eight years; male; Mexican; schoolboy.

Family History.—Mother and one brother show Abel.

Past History.—Very large crusts in nose, which are hard to remove. Very foul odor, but more typical of the Friedlander type.

Bacteriologic Examination.—Abel bacillus.

Examination.—Nose completely blocked with large black crusts, which are extremely hard to remove. They are very hard and dry.

Treatment.—January 8, 1916. Injection stock Perez, 100 million.

January 22, 1916. Injection stock, 200 million. No reaction.

January 27, 1916. Injection stock No. 3, 500 million.

January 29, 1916. Injection autogenous vaccine, 250 million. No reaction from last injection. Crusts are more moist and easier to remove.

February 1, 1916. Injection autogenous vaccine, 500 million. Crusts the same; no odor.

February 5, 1916. Odor and crusts worse. Injection autogenous vaccine, 1 billion.

February 15, 1916. Injection autogenous vaccine, 1 billion. No odor; crusts decidedly improved. No reaction from last injection.

Note.—This is the typical bacillus mucosus capsulatus of the large plump type. The treatment, both in this case and his brother and mother, both of whom had exactly the same infection, has been very unsatisfactory. The cases have been very irregular in attendance. The use of the autogenous vaccines in this case was an experiment of little value. Patient since above dates has been taking Perez stock vaccines, with marked improvement.

Case 25.—M. C., aged fourteen years; female; Chinese; schoolgirl.

Family History.—Negative.

Past History.—Very indefinite.

Bacteriologic Examination.—Friedlander.

Examination.—Odor very strong and perceptible at three feet. Marked atrophic rhinitis. Crusts entirely blocking the nose.

Treatment.—March 16, 1916. Injection Perez stock No. 6, 200 million.

March 17, 1916. Injection No. 6 stock, 400 million. Condition about the same.

March 18, 1916. Injection No. 6 stock, 1 billion. Nose entirely free from crusts. No odor. No reaction.

March 20, 1916. Injection No. 6 stock, 1½ billion. No crusts or odor.

March 24, 1916. Injection No. 6 stock, 2 billion. No crusts or odor. No reaction.

March 28, 1916. Injection No. 6 stock, 3 billion. Condition same.

March 31, 1916. Injection No. 6 stock, 3 billion. Nose perfectly free from crusts and odor. No reaction.

April 5, 1916. Injection No. 6 stock, 1 billion. No crusts or odor. No reaction.

April 8, 1916. Injection No. 6 stock, 3 billion. No crusts or odor. No reaction.

Note.—This case is one of the most brilliant improvements in the Abel group. The crusts cleared up quickly and remained so. The odor has not returned since the third injection. Case under treatment.

Case 26.—W. B., aged twenty-seven years; female; English.

Family History.—Negative.

Past History.—When seven years of age noticed trouble. Has treated more or less; no improvement. Used to play with dogs. Has lost sense of smell.

Bacteriologic Examination.—Friedlander.

Examination.—Typical atrophic rhinitis of true ozena. It is impossible to make out the turbinates. Crusts plaster the entire inside of nose. Odor foul and heavy. Wassermann negative.

Treatment.—February 25, 1916. Injection No. 5 Perez stock, 200 million.

February 26, 1916. Injection No. 5 stock, 400 million. No reaction.

February 28, 1916. Injection No. 5 stock, 500 million. Crusts and odor about the same. No reaction.

March 3, 1916. Injection No. 5 stock, 1 billion. Patient feels decidedly better.

March 6, 1916. Injection No. 5 stock, 2 billion. No headaches, crusts or odor.

March 10, 1916. Injection No. 5 stock, 2 billion. General reaction. Malaise.

March 15, 1916. Injection No. 5 stock, 2½ billion. Slight reaction. No crusts or odor.

March 20, 1916. Injection No. 5 stock, 2 billion. Severe reaction one hour after injection. Nausea, headache, lasting two days. Nose feels better. No odor. One large crust on left size.

March 24, 1916. Injection No. 6 stock, 2 billion.

March 27, 1916. Injection No. 6 stock, 3 billion.

April 5, 1916. Injection No. 6 stock, 3 billion. Slight whitish crusts on both sides. Slight odor on breathing directly into face. No reaction.

April 8, 1916. Injection No. 6 stock, 4 billion. No crusts or odor.

Note.—This case has been under treatment for years in various clinics. She has received many injections of various types of vaccines. There is an infection of the left sphenoid and ethmoid with causes constant secretion and crusting in the left side. The injections have given the patient more satisfaction than any previous treatment, and the odor is certainly decidedly better.

Case 27.—V. M., aged thirteen years; female; American; schoolgirl.

Family History.—Negative.

Past History.—Mother noticed ozena trouble since child was four years old. Last three or four years odor remains the same. Odor worse at times.

Bacteriologic Examination.—Friedlander, Abel.

Examination.—Marked atrophic rhinitis with thin white crusts spreading over turbinate, septum and floor of nose. Nose washed twice a day.

Treatment.—November 23, 1915. Injection Perez stock, 250 million. Nose not washed for one week. Slight ozena odor. Right nostril entirely blocked with blackish crusts. Left filled with whitish crusts. Mother says that odor is not as bad now as it gets to be.

November 26, 1915. General reaction, headache, nausea and dizziness. Local reaction. Crusts and odor less.

December 8, 1915. Injection stock, 250 million. Nose not washed for one day. Large typical crust on left side; very slight characteristic odor of ozena. Patient says that odor is improving. Crusts very hard to remove. Patient generally looks very much improved.

December 15, 1915. Injection stock No. 3, 500 million. Condition about the same.

December 29, 1915. Injection stock No. 3, 250 million. General reaction immediately following last injection. Nausea, headache, chills, fever and dizzy. Reaction lasted three days. Patient noticed that within two days from injection crusts practically disappeared, and they are much easier to blow out. Examination: Absolutely no odor; few crusts far back.

January 5, 1916. Injection Sherman's No. 36. Entire nose filled with crusts. Condition about the same as in first examination. No reaction. Odor strong, but not typical of ozena.

January 8, 1916. Injection autogenous vaccine, 300 million. Slight odor. No crusts.

January 10, 1916. Injection autogenous vaccine, 600 million. Slight nose bleed from last injection. Slight odor. Crusts easier to remove. No reaction.

January 15, 1916. Injection autogenous vaccine, 600 million. No crusts or odor. No reaction.

January 21, 1916. Injection autogenous vaccine, 1 billion. Condition same. No reaction.

January 26, 1916. Injection autogenous vaccine, 1 billion. Odor same. Few crusts. Patient notices great improvement.

January 31, 1916. Injection autogenous vaccine, 1 billion. Crusts much easier to remove. No odor. Has not washed nose for twenty-four hours. No reaction.

February 4, 1916. Has not washed nose for two days. No odor; few crusts. Treatment stopped for two weeks. Local reaction.

February 10, 1916. No odor; slight amount of crusts.

February 19, 1916. Injection No. 5 stock, 100 million. Crusts decidedly improved. No odor.

February 23, 1916. Injection No. 5 stock, 200 million. Nose not washed for one day. Interior plastered with whitish crusts. No odor. In spite of presence of the crusts, patient does not notice them, and they are not so difficult to remove.

Note.—This case was placed in the Friedlander group. The improvement while under treatment was remarkable, but often in three or four days the nose would fill up as noted on January 5th, and it would seem that no improvement had taken place. The patient was very stubborn and hard to handle, and finally refused further treatment,

RABBIT EXPERIMENTS.

The pathologic changes in the nose of rabbits, following the intravenous injections of cultures of the Perez bacillus, have been carefully investigated by Hofer[113] and more recently by Burckhardt and Oppikofer[143]. The local manifestations in the turbinates following one or more injections are marked. Our specimens show the whole gamut of changes from the intense congestion following a fatal dose to a complete atrophy with actual destruction of the bone.* The observations of Burckhardt and Oppikofer were carefully and painstakingly made, but their negative results and findings are so at variance with our own and the findings of Hofer and Perez, that we can only attribute them to the use of cultures so often subcultured that their virulence had been greatly lowered.

Our interest in the matter, however, has been chiefly in methods of producing rapid agglutinating sera, determination of toxicity, and fatal dose. It is not our purpose to criticise the paper of Burckhardt and Oppikofer. Our rabbit protocols are not repeated here in detail. (See Chart No. 6.) We do, however, feel that working with but two subcultures furnished by Hofer, their results are bound to be unsatisfactory and inconclusive. Our work, as shown in Chart No. 6, included fifteen strains. The most unsatisfactory results were obtained with cultures furnished by Hofer. These were marked originally I and II, but whether they were the same cultures furnished Burckhardt and Oppikofer, also marked I and II, we are unable to state. It was only after animal passage that we were able to obtain satisfactory results with these strains.

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*Details of histopathologic studies have been purposely omitted and will be presented in our forthcoming paper.

As a rough basis for the estimation of the dose, to a fresh vigorous twenty-four hour agar transfer, five cubic centimeters of salt solution was added, and an emulsion made, as shown in Chart No. 6. The rabbits injected with the killed cultures, following the method of Hofer, lost flesh very rapidly, and it seemed impossible to secure an agglutinating serum. A dose of one and one-half to two cubic centimeters (Chart No. 6, Rabbits Nos. 1 to 10) seemed invariably fatal. The changes in the nose were marked, but we could not isolate the Perez from the nasal secretion or use the sera for agglutinating purposes.

As soon as live cultures were used (Nos. 11 to 49), the rabbits held their weight better, an agglutinating serum was easy to obtain, and some of the rabbits lived as long as three months. One animal lived nearly six months, but the average length of life is two to three months, after three to five injections of a maximum dose of two cubic centimeters.

We have recently found that if the dose is started very small and repeated at frequent intervals, a serum agglutinating as high as 1 to 2,000 can be obtained in a few weeks. (Nos. 38 to 43.) It is well to try the agglutinating power after the fourth injection, and if present to keep the rabbit without further injection, or allow at least a ten day interval to elapse. In this way, if all conditions are favorable, the animals can be kept alive for six months.

Experiments are now under way to determine the endo- and exo-toxin content, and to try and explain the great variability in the toxicity of the various strains. A study of Chart No. 6 will prove useful in determining the maximum dosage. The details are self-explanatory. Full autopsy protocols are not given here, as they shed no light on the question of dosage.

We cannot understand the inability of Burckhardt and Oppikofer to demonstrate the Perez bacillus in the nasal secretion of injected rabbits. In spite of most careful search, they failed in every case.

As shown in Chart No. 6, the organism was recovered, in many cases, in pure culture and proved by agglutination. The typical coryza came on after the first injection, and in two cases, small healthy rabbits, previously shown to be free from Perez, contracted acute coryza by cage contact with the diseased animals, and the Perez was demonstrated in pure culture from their nasal secretions.

We were also able to demonstrate the pure culture from the heart, blood and lungs, the latter habitat being especially significant in the light of our observations concerning the relation of the Perez bacillus to the bacillus bronchisepticus of dogs.

The statement that they were never able to produce atrophy of the turbinates is directly at variance with our own experience. Our specimens show many such cases. Their claim that the virulence of the culture was proved because the animals died of acute septicemia with large doses, is open to serious objection. The animals probably died from acute toxemia which follows the administration of large doses.

Their contention, that the absence of crusts and odor in the nose of rabbits is an important point against the specificity of the Perez infection, is unconvincing. Crusts, per se, have nothing to do with ozena. The formation of crusts depends on the chemical composition of the secretion. The odor is always less marked when the secretion acquires a watery character. In our own rabbit experiments, where ten to fifteen animals were confined in one cage, a very characteristic odor could be noted ten to fifteen feet from the cage. In certain animals where the process was far advanced, a characteristic ozena odor was noted as soon as the head was split open. Their admission, that they never saw atrophy in their animals, is the best proof that they were not dealing with advanced cases.

BACTERIOLOGIC STUDIES.

This part of the paper embraces a more intensive biologic study of "coccobacillus fœtidus ozænæ" (Perez) than contained in the previous description of Hofer[113], and comparative studies of Bacillus bronchisepticus and Bacillus mucosus capsulatus. In some instances we are at variance with Hofer concerning the morphologic and metabolic behavior of the organism. Bacillus bronchisepticus, exhaustively studied by Ferry[140], McGowan[141], Torrey and Rahe[142], and determined by them as being the specific organism of canine distemper, morphologically almost identical and biologically in many instances similar to the Perez organism, and the advanced hypothesis that the infection of fetid ozena is carried by dogs, presents an interesting complex. The frequency with which organisms of the Friedlander group are isolated from typical and atypical ozena cases, biologic comparatives, changes clinical and of the nasal bacterial flora under immunologic measures with bacillus Perez vaccin, led us for the time being to the consideration of a possible bacterial mutation. Burckhardt and Oppikofer[148] have placed the organism tentatively among the Friedlander-aerogenes group. By our own and other studies we are inclined to doubt this contention, and tend rather to place the organism with the colityphoid group. Some bacteriologists may be inclined to place the organism with the hemorrhagic septicemia group.

Morphology.—The vegetative cell from twenty-four-hour agar cultures falls into one of two groups: (1) A distinct coccoid rod with a poorer staining protoplasmic equator, though not sufficient to confuse as a diplococcus. The ends are round. Dimensions from $0.75—1.25 \times 0.4$ microns. (2) A larger and thicker bacillus which stains more uniformly. Ends less rounded. Dimensions, $1.0—1.5 \times 0.4—0.6$ microns. A relative constancy in the sizes of young culture organisms is encountered; pleomorphism, as described by Hofer, appearing only in old cultures. The bacillus from young broth cultures has no distinction from cultures from solid media. In old broth cultures the organism is larger and pleomorphism is marked. Chain formation, except for occasionally two or three elements, never occurs.* The bacillus is uniformly Gram negative. Stains with ordinary dyes, though not intensely. Bipolar intensity in coccoid type. Spores or capsule are never developed.

Agar Plate Colonies.—In twenty-four hours at 37° C., surface plate cultures are plainly visible. Round, slightly elevated colonies, one to two millimeters in diameter. Edge entire. Translucent and lustrous by reflected light, bluish by transmitted light. In forty-eight hours colonies are doubled in size, circular or slightly ameboid, slightly elevated, flat or slightly convex. Edge undulate. The colony becomes slightly less transparent and somewhat whitish. Microscopically the colony is of high refractile index and internal structure homogeneous, later becoming finely granular; center grumose in old colonies. Old colonies show little variation except for increased size to about eight millimeters in diameter, central opalescence and lessening of luster. Blueness by transmitted light of the less dense periphery prevails.

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*Long chain formation with bacillus bronchisepticus is the rule; in young broth cultures appearing in hanging, drop-like, long slender rods.

Agar Stroke.—In twenty-four hours at 37° C., a luxuriant growth has developed along line of stroke. Flat, with transparency increasing toward edges. Edge slightly elevated, lobate-lobulate, microscopically showing marginal colony development (lappenbildung). In most instances the medium ultimately becomes deeper yellow or brownish in color—one strain producing an oxidization of the medium to an intense brown.

Plain Broth.—In twenty-four hours there is produced a diffused turbidity of the medium. No change for several days, when a granular deposit develops. There is no scum formation. The medium gives off a sickening odor, but is uncharacteristic.

Motility.—In our experiences, the organism from twenty-four-hour broth cultures is definitely motile in all instances: with certain strains a typhoid-like activity is manifest, but usually the hanging drop shows definite but slow motility with marked activity of occasional organisms.* In older broth cultures (seven days), motility persists, though weaker. Long flagella—usually six are demonstrable.

Gelatin Stab.—In forty-eight hours a delicate filiform growth has developed along line of stab. There is nothing characteristic in older cultures. Gelatin is not liquefied.

Potato.—In twenty-four hours at 37° C., a moist yellowish discoloration of the surface of the medium appears. The outline of the growth is not usually definite. With certain strains the growth may be colorless or slightly whitish. There is no unusual discoloration of the potato, or it may be slightly discolored brown. In forty-eight or seventy-two hours the growth becomes more distinct and raised. The surface becomes uneven and coppery in color. Medium discolored.

Litmus (Azolitmin) Milk.—Certain strains show an increased alkalinity or no change in the reaction of the medium, while the greater number show a slight reduction of the litmus, and some produce definite acid production with coagulum.* (See Table 7.) Acid production is slow, and rarely occurs before forty-eight or seventy-two hours. Cultures of various ages show some inconstancy in reaction.

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*Hofer described the organism as being immotile. This same characteristic is ascribed by Burckhardt and Oppikofer, although in only one of three cultures submitted to them by Hofer was the organism found motile, this characteristic prevailing through animal experiments.

Endofuchsin Agar.—In the first twenty-four or forty-eight hours the colonies are apparently nonacid. After this time the centers of the colonies become red, while the periphery continues white.

TABLE No. 7

Carbohydrate Fermentation and Reaction Record of Various Coccobacillus Ozena Fetidus Cultures.

Culture	Glucose		Lactose		Malt.	Mann.	Sacc.	Litmus Milk		
	G%	A%	G%	A%	G%	G%	G%	1 day	2 day	7 day
Adolphus	–		–		–	–	–	–	–	Lilac
Femol	S1		S1		–	–	–	–	–	A+C
Kelleher	12	3.2	–	1.7	5%	10	10	A+C	A+C	A+C
Strum	–		–		–	–	–	–	–	–
Piffle	–		–		–	–	–	A+C	A+C	A+C
Plant	–		–		–	–	–	–	Alk.	Alk.
Weber	–		–		–	–	–	–	Lil.	A
S. L.	–	1.3	–	1.5	–	–	–	–	A+C	A+C
Mrs. Miranda	10	2.6	–	.05 Alk.	S1	10	10	–	Lil.	A
Vienna		2.8		.7				–	Alk.	Alk.
Stefanovich								–	–	Lilac
Spencer	10		–					–	–	–
Bloomenberg	S1	.6		.2 Alk.				–	–	–
18821	S1	1.9		.7 Alk.				Lil.	A+C	A+C
W. B.	12							–	–	Lilac
H. Miranda,	10		–					–	–	–

(Gas and acid readings are after forty-eight hour incubation.)

Carbohydrate Fermentation.—There is nothing consistent in carbohydrate fermenting capabilities. Culture age gives variation. Some cultures are ineffective with all the carbohydrates. Dextrose is most often fermented (bubble to fifteen per cent). Lactose is never effected. Maltose, mannite and saccharose are variably and indifferently fermented. (See Table No. 7.)

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*Hofer states litmus milk is unchanged. Burckhardt and Oppikofer state that acidity is occasionally produced, but that milk is never coagulated.

Acid Production in Dextrose and Lactose Broth.—Reaction change in dextrose broth is usually acid. In twenty-four hours the change may be from none to three per cent acidity. In the next twenty-four hours there may be but little change, but the acidity percentage is most frequently reduced. In seven days little or no change in the reaction takes place.

No appreciable change is observed in twenty-four-hour lactose broth cultures. In forty-eight hours the acidity may reach one per cent, but the change to alkalinity is usual. In seven days little or no change from the forty-eight-hour reading is observed. (See Table No. 7.)

Reduction of Nitrates to Nitrites.—Like bacillus bronchisepticus, the Perez organism can be broadly divided into two groups, according to its nitrifying ability. Of the organisms isolated by us they were almost equally divided in this respect.

Anaerobiasis.—Aerobic, but readily facultates to anaerobic. Growth persists in deep and surface anaerobic cultures. In fermentation tubes both arms are at first uniformly turbid, with increasing turbidity in the open arm.

Odor.—We are unable to detect a marked similarity in the nasal odor of ozena patients and cultures, although culture odor is offensive.* It is not dissimilar to certain other bacterial cultures.

The salient morphologic and biochemic characteristics of the Perez bacillus are: A short, Gram negative, motile coccobacillus, showing more intensive polar staining. It is never capsulated, and spores are not borne. Gelatin is not liquefied. Growth is never mucoid. Agar colonies are somewhat colilike. It may or may not reduce nitrates to nitrites. It is not fastidious in the fermentation of carbohydrats or in the production of acid. Growth on potato is fairly characteristic, although not constant. Odor from broth cultures is offensive, but not characteristic. It may simulate certain of the Abel's bacillus of the Friedlander group, and at times render confusion in morphology and certain biologic characteristics. The Abel bacillus, though, can be made to develop capsule and is never motile. With the most painstaking effort we were unable to develop a capsule with the Perez organism. In size, shape, tinctoral reaction and motility the bacillus bronchisepticus and bacillus Perez are almost identical, although the former is slightly heavier. Grouping according to nitrification is permitted of both organisms. Growth upon potato is similar in most instances. Bacillus bronchisepticus tends to grow in chain formation in broth, is almost strictly aerobic, tends to alkali production and splits no carbohydrats. Bacillus Perez generally differs in all these respects. (Table No. 8.)

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*Hofer has maintained that young cultures, especially broth, have an odor similar to the characteristic odor of ozena in man.

In passing, we wish to place emphasis upon motility and frequent acid production, as demonstrated by us, in contravention to the observations recorded in previous publications. From a culture brought by one of us from Hofer's laboratory the organism was proven to be definitely motile. Likewise do we find relatively frequent tendency to acid production with certain cultures, although the medium reaction is unchanged in many instances, and occasionally an increased alkalinity develops.

Agglutination Studies.—A potent agglutinating serum was easily produced by immunization of rabbits with living organisms. A high agglutination titre, 1:1000 to 2000, existed for homologous strains, a relatively lower titre existed for allied strains. For group identification of the organism we found that low serum dilutions (1:160) were sufficient for practical purposes. Even in this dilution strains could be coarsely separated. (Table No. 9.) The agglutinability of the organism is always low until after a suitable laboratory life.

Abel's bacillus and others of the Friedlander group were never agglutinable, even in very low dilutions (1:20). Two strains of bacillus bronchisepticus were non-agglutinable with antisera—Rabbit No. 3 (Adolphus)—the only agglutination experiment carried out with this organism; yet this organism proved to be markedly antigenetic in a complement deviation test with this antisera.

Complement Fixation Studies.—Several complement fixation experiments were carried out for bacterial classification by specific protein determinations, using a Perez antiserum and filtered autolysates of selected Perez, Abel and bronchisepticus cultures. The results of these experiments were striking and conclusive. After determining a constant and definite antigenic power of the Perez autolysate with various immune sera, the antigenic power of the Abel and bronchisepticus autolysates with Perez antisera was studied. The Abel antigen was

TABLE NO. 8.

Comparison Between B. Bronchisepticus and B. Perez,

	B. Bronchisepticus	B. Perez
Morphology	Two forms—short coccobacillus and larger type. 0.75—1.25x0.4—0.5 microns 1.00—1.6 x0.4—0.5 "	Same 0.75—1.25x0.4 and 1.00—1.5x0.4—0.6 microns
Staining	Not intense. More at poles, especially coccoid type. Gram negative.	Same
Motility.	Feebly motile.	Same Occasionally active.
Gelatin.	Not liquefied.	Same.
Reduction of Nitrats to Nitrits	Divided into negative and positive group.	Same.
Agar Colony.	Delicate coli-like. Usually not visible in twenty-four hours.	Similar. Usually visible in twenty-four hours.
Broth Cultures.	Turbid—often scum. Long chain formation.	Turbidity. Never scum. Never in chain formation.
Potato.	Yellow, coppery colored growth. Potato discolored brown.	Usually indistinguishable. Occasionally colorless.
Litmus Milk.	Surface alkalinity.	Frequent acid production. Milk may coagulate. May be no change or alkalin.
Carbohydrats.	No ferment or acid.	Dextrose frequently fermented, with production of acid. Lactose and other sugars rarely or slightly effected.

Anaerobiasis.	Aerobic. Slightly anaerobic.	Grows under aerobic or anaerobic conditions, but preferably aerobic.
Rabbit Inoculation.	Dead culture highly toxic. Living culture better tolerated. Bacterial infarcts without suppuration. Agglutinating serum easily established. Wasting of animal.	Same.
Agglutination	Easily by bacillus bronchisepticus antisera. Not by Perez antisera.	Easily agglutinated by Perez antisera. Bacillus bronchisepticus antisera not attempted.
Complement Fixation.	Autolysate as antigen produces complete deviation of complement with bacillus Perez antisera.	Same.

impotent, and no fixation of complement was brought about. The bronchisepticus autolysate, on the other hand, was equally as active as the Perez antigen in complement binding. Complement fixation tests with serum of clinical and bacteriologic ozena cases were always negative.

TABLE No. 9

Agglutination Record for Group Identification of Coccobacillus Ozenae Fetidus Cultures and Coarse Separation of Strains. Group Differentiation of Abel's Bacillus and B. Bronchisepticus.

Culture	Rabbit Antisera							
	No. 1 (Vienna)	No. 23 (Alpert)	No. 5 (Plant)	No. 7 (Kelleher)	No. 3 (Adolphus)	No. 11 (Femol)	No. 25 (18821)	No. 27 (Piffle)
B. Perez,								
Adolphus	+	+	+	+	+	+	+	
Alpert	+	+	+	−	+	−	+	
Femol	+	+	+	−	−	+	+	
Kelleher	−	−	−	+	−	−	−	
Strum	+	+	+	+	+	+	+	
Griffin	+	+	+	+	+	+	+	
18821	+	+	+	+	+	−	+	
Piffle	+	+	−	−	−	+	+	+
Mrs. Miranda	+	−	−	+	+	−	+	
Vienna	+	+		+	+	+	+	
Rab. 11 Femol	−		−	+	+	+		
Rab. 8 Vienna	+		+					
Weber	−	−	−	−	−	+	+	
Kelleher B.	+				+			
Plant	+		+					
S. L.	+				+	+		
Stefanovich	+							
Spencer					+			
B. Abel's,								
Knealy	−		−	−	−	−	−	
Grove	−			−	−	−	−	
Tuckey	−		−	−	−	−		
Faber	−		−	−			−	
B. bronchisepticus,								
No. 1					−			
No. 2					−			

Animal Observation.—Immunologic measures with killed cultures were found unsatisfactory, owing to a marked toxicity of killed cultures and the difficulty of procuring an agglutinating serum of sufficient titre, and were early abandoned. No studies were carried out to determine the toxicity of bacterial filtrates, but the toxic fraction is apparently very high.

The animals (rabbits) enjoyed a much greater tolerance to living cultures, and the immunity response was marked. Under cultural injections (intravenous) the animals rapidly lost in weight, and ultimately succumbed to intercurrent infections.

The changes in the nose are described elsewhere in this paper.

Under injections of large doses, bacterial infarcts formed in various portions of the body. These did not go on to suppuration. Not infrequently such infarcts developed on the ears, and cultures from these showed viability after many months.

The organism was isolated from the heart blood, nose and lungs. In one instance we were able to isolate the organism from the nose of another animal that had received no injections, but had been caged with an injected animal.

Experimental inoculations by submucous injection were positive in that the organism was recovered with associated atrophic changes.

Clinical Bacteriologic Considerations.—From forty cases of typical ozena under observation the Perez organism was recovered in twenty-eight instances. In not a few instances we were compelled to make repeated attempts before the organism could be isolated. Isolation of the organism is beset with no difficulties except when the accompanying bacterial flora contains weedy overgrowing types. The commonest offenders are the bacillus pyocyaneus and organisms of the Friedlander group.

The Abels bacillus and others of the Friedlander group are deserving of special consideration. Of the forty cases studied by us, some of the Friedlander group, usually Abel's, were isolated in twelve instances to the exclusion of positive Perez findings. In five instances of an initial positive Perez diagnosis Friedlander organisms appeared in cultures after Perez vaccine therapy with negative Perez cultures. In one instance (Kelleher) Abel's and Perez bacilli were easily isolated from the same culture.

Our search for Perez in plates giving colonies of the Abel's bacilli was not as thorough as it should have been, owing to the belief, for a considerable time entertained by us, that a bacterial separation of fetid ozena cases might be made, or that the organisms were coetiologic. In the light of recent studies we are inclining to the belief that the activities of the Perez bacilli in the nose creates a condition symbiotic for Friedlander organisms, and that negative Perez findings from typical cases were due to technic difficulties. The belief is now entertained by us, though not yet verified, that through the activities of the Perez bacillus there is created an environment favorable for the development of organisms of the Friedlander group, and these in turn exercise an action antagonistic to the Perez organism. There are many parallels for the assumption of this attribute of the Friedlander bacilli.

In a considerable series of studies of the nasal bacterial flora of nonozena cases, the Perez coccobacillus was never found, except in three cases of syphilitic ozena. These cases, though, were typical ozena. Organisms of the Friedlander group were not infrequently present.

Again, Abels bacilli vaccins are without the slightest influence in Abels ozena cases, but the focal response in these cases under Perez vaccin therapy is startling.

The theory that dogs are the medium of fetid ozena transmission has been both exploited and questioned. Up to the present time we have given no consideration to this phase of ozena, and have taken bacillus bronchisepticus for comparative study, as it is the organisms more like coccobacillus ozenæ fetidus than any other of which we have knowledge. Distemper, a common and often an epidemic canine malady, begins as a nasal infection. While kennels may at times be decimated, the animals usually recover. Infection produces prostration, but death is usually due to secondary invaders. Distemper carriers are not common.

Résumé.—Some characteristics of coccobacillus fetidus ozenæ (Perez) have been heretofore erroneously described, in that the organism is motile, may ferment carbohydrats and may produce acid.

Coccobacillus ozenæ fetidus and Abel's bacillus are distinct bacterial entities.

There is a startling similarity between coccobacillus ozenæ fetidus and bacillus bronchisepticus, the specific organism of canine distemper. This similarity is pronounced in complement fixing phenomenon.

The coccobacillus fetidus ozenæ group is made up of many subvarieties or strains.

Coccobacillus fetidus ozenæ is probably the specific organism of typical fetid ozena in man.

A change in the name of the organism from coccobacillus ozenæ fetidus to bacillus rhinosepticus is proposed.

GENERAL CONCLUSIONS.

The conclusions stated in our preliminary report, with exceptions about to be noted, still stand, and we repeat them here:

1. The coccobacillus fetidus ozenæ Perez, as isolated by Hofer, has answered all the bacteriologic requirements necessary to establish its identity as the etiologic factor in ozena.

(Note.—We feel that this conclusion must be accepted with certain reservations. We have been unable to obtain a complement fixation with untreated ozena patients. Whether this failure is due to faulty technic or, for various reasons, impossible of accomplishment, we are unable to state.)

2. The isolation of this organism is attended with considerable difficulty.

(Note.—This statement no longer holds, as with our discovery of the motility of the organism, our improved methods of isolation, and the ease with which the cultures can be proved by agglutination and complement fixation reactions, the matter is comparatively a simple one.)

3. The production of agglutinating serum in rabbits is an exceedingly difficult task.

(Note.—The use of live instead of killed cultures has enabled us with as few as four injections, and within a period of four weeks, to produce a serum which agglutinates in a dilution as high as one to two thousand.

4. The preparation of autogenous vaccins in every case is very difficult, if not impossible.

(Note.—This statement refers to the difficulty of isolating the Perez organism in the Friedlander group. Now that we have found that almost as brilliant results can be obtained in this group by means of our stock Perez vaccin, this difficulty in the treatment has been overcome.

5. At present mixed vaccins made from various strains of

Perez bacillus is the most practical method of treatment which is now available.

(Note.—This statement stands without change.)

6. It may be necessary to precede or combine with the treatment the vaccins made from the organisms which are usually present in combination with the Perez bacillus.

(Note.—As already noted, the preliminary treatment with a mixed stock vaccin has proved unnecessary. In our first vaccins we combined the Perez bacillus with the Friedlander, micrococcus catarrhalis, pneumococcus, and other well-known organisms found in chronic catarrhal conditions, and found very quickly that these mixed vaccins were not nearly as efficacious as vaccins made from the various strains of the Perez organism.

7. It may be possible that there may be two or more types of ozena, bacteriologically different but clinically identical.

8. In spite of the presence of organisms of the Friedlander group, in cases of undoubted clinical ozena, we consider that these organisms have no etiologic significance.

(Note.—As already noted, we have, for the sake of convenience, divided clinical ozena into two bacteriologic groups. With improved technic and further morphologic studies, we believe the socalled Friedlander group will become increasingly smaller.)

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CHART I.

Bacteriologic Findings in Cases of Rhinitis Atrophicans Nonfetida.

No.	Name	Age	Sex	Nationality	Referred by	Clinical Diagnosis	Bacteriologic Examination	Odor	Crusts	Notes.
1	C. K.	18	M.	Jew	Office	Antrum and frontal sinus suppuration	Staphylococci	O	O	No improvement with autog. vaccin.
2	C. W.	20	M.	American	Poly.	Rhinitis Atrophicans	Staphylococci	O	O	
3	V. D.	35	F.	American	Poly.	Rhinitis Atrophicans	Staphylococci	O	O	Empyema antrum.
4	E. U.	25	F.	American	Poly.	Rhinitis Atrophicans	Staphylococci	O	+	
5	S. T.	27	F.	German	Poly.	Rhinitis Atrophicans	Staphylococci	O	O	
6	S. C.	26	F.	American	Nise	Rhinitis Atrophicans	Pneumococci	+	+	Case reported improved with autog. vaccin.
7	O. O.	?	?	American	Lewitt	Rhinitis Atrophicans	Micrococcus catarrhalis	+	+	Odor not typical.
8	McG.	31	F.	American	Poly.	Rhinitis Atrophicans	B. pyocyaneus	O	O	

9	G. M.	6	M.	American	Poly.	Rhinitis Atrophicans	Pneumococcus	0	0	
10	M. J.	30	M.	American	U. C.	Normal Mucous Membrane	Staphylococci	0	0	Father of case 30, Chart II. Congenital syphilis.
11	F. J.	5	F.	American	U. C.	Normal Mucous Membrane	Staphylococci	0	0	Daughter of M. J.
12	B. J.	6 mos.	F.	American	U. C.	Normal Mucous Membrane	Staphylococci	0	0	Daughter of M. J.
13	E. J.	30	F.	American	U. C.	Normal Mucous Membrane	Staphylococci	0	0	Wife of M. J.
14	J. G.	50	M.	French	Office	Rhinitis Atrophicans	Micro-catarrhalis	0	0	
15	H. G.	54	M.	American	Office	Rhinitis Atrophicans	Staphylococci	0	0	

CHART II.

Perez Group. All Cases True Clinical Ozena. General Analysis.

No.	Name	Age	Sex	Nationality	Referred by	Clinical Diagnosis	Bacteriologic Finding	Agglutination	No. of Inject's	Crusts	Odor	Result.
1	Adolphus	28	M.	German	Victors	Ozena	Perez	+	20	+	+	No crusts, no odor. Clinical cure. Under treatment.
2	Alpert	22	M.	Russian Jew	Richards	Ozena	Perez	+	10	+	+	No crusts, no odor. Clinical cure.
3	Evadikin	25	F.	Russian Jew	Hutchins	Ozena	Perez	+	10	+	+	Not treated by me. Reported improvement marked.
4	Femol	16	M.	?	Hunt	Ozena	Perez	+	?	+	+	Not treated by me. No report to date.
5	Faber	33	M.	French	Poly.	Ozena	Perez	+	6	O	+	No crusts, no odor. Clinical cure, patient's standpoint.
6	Griffin	21	F.	American	Green	Ozena	Perez	+	15	+	+	No crusts, no odor. Clinical cure.
7	Hillman	21	F.	American	Office	Ozena	Perez	1	15	+	+	No crusts, no odor. Clinical cure.

8	Kelleher	17	F.	German	Victors	Ozena	Perez	+	20	+	+	No crusts, no odor. Clinical cure. Under treatment.
9	Luoto	40	M.	German	Stanford	Ozena	Perez	+	7	O	+	No crusts, no odor. Under treatment.
10	Miranda, Mrs.	37	F.	Spanish	Poly.	Ozena	Perez	+	9	+	+	No crusts, no odor. Under treatment.
11	Miranda, J.	11	M.	Spanish	Poly.	Ozena	Perez		9	+	+	No crusts, no odor. Under treatment.
12	Miranda, H.	12	M.	Spanish	Poly.	Ozena	Perez		9	+	+	No crusts, no odor. Under treatment.
13	Novani	14	F.	American	Office	Ozena	Perez		12	+	+	No crusts, slight odor. Stopped treatment.
14	Plant	20	F.	American	Office	Ozena	Perez	+	19	+	+	No crusts, no odor. Clinical cure.
15	Piffle	30	M.	American	State Insane Asylum	Ozena	Perez	+		+	+	No treatment.
16	Spencer	?	M.	?	Spencer	Ozena	Perez	+		+	+	No treatment.
17	Stefanovich	25	M.	Servian	Stanford	Ozena	Perez		10	+	+	No crusts, no odor. Clinical cure.

CHART II. (Continued)

Perez Group. All Cases True Clinical Ozena. General Analysis.

No.	Name	Age	Sex	Nationality	Referred by	Clinical Diagnosis	Bacteriologic Finding	Agglutination	No. of Inject's	Crusts	Odor	Result.
18	S. L.	16	F.	American	Johnson	Ozena	Perez	+		+	+	Meagre reports, great improvement. Not seen by me.
19	Strum	19	F.	American	Hutchins	Ozena	Perez	+	10	+	+	Some crusts, some odor. Great! improvement. Not seen by me.
20	Spooner	35	M.	American	Office	At Rhin.	Perez	—	5	O	O	Ozera larynx.? Great improvement.
21	Pris	22	F.	German	Office	Pan Sinusitis	Perez	—	19	+	+	No crusts, no odor. Clinical cure.
22	Weber	27	M.	American	Office	Ozena	Perez	+	14	+	+	No crusts, no odor. Clinical cure.
23	Zamla	25	F.	Hungary	Office	Ozena	Perez		3	+	+	Marked improvement. Did not return.

24	De Vecci	18	F.	Italian	Poly.	Ozena	Perez			+	+	No treatment.
25	Spicer	?	?	?	Spicer	Ozena	Staph			+	+	No treatment.
26	Smith	22	F.	American	Benedict	Ozena	Staph			+	+	No treatment.
27	C. K.	13	M.	American	Maxey	Ozena	Staph			+	+	No treatment.
28	Jolley	30	M.	Russian Jew	Poly.	Ozena	Staph		7	+	+	No crust, no odor. Clinical cure, patients standpoint.
29	Morgan	21	F.	American	Stanford	Ozena	Perez		4	+	+	No crust, no odor. Clinical cure, patients standpoint.
30	Jackson	9	F.	American	U. C.	Ozena	Perez	+	O	+	+	Congenital syphilis. No treatment.
31	J. W.	50	M.	American	U. C.	Ozena	Perez	+		+	+	Active syphilis. No treatment.
32	E. H.	37	M.	American	U. C.	Ozena	Perez	—		+	+	Active syphilis. No treatment.

CHART III.

Clinical Ozena. Friedlander Group. Treated with Perez Vaccine. General Analysis.

No.	Name	Age	Sex	Nationality	Referred by	Clinical Diagnosis	Bacteriologic Examination	Odor	Crust	No. of Injections	Agglutination with Perez Strains	
1	Walsh	23	F.	American	Poly.	Rhinitis atrophicans nonfetida	Friedlander	O	O			
2	Rosberg	18	F.	American	Poly.	Rhinitis atrophicans nonfetida	Friedlander	O	O			
3	Ticox	22	M.	American	Nagner	Ozena	Friedlander	?	+			
4	Tuckey	30	M.	American	Office	Rhin. Atroph.	Friedlander	O	O			Several examinations showed organism closely resembling Perez.
5	Ortereras	30	F.	Mexican	Poly.	Rhin. Atroph.	Friedlander	O	O		Neg.	
6	Kenealy	35	F.	American	Office	Rhin. Atroph.	Friedlander	O	+		Neg.	History of typical ozena in childhood. No odor for years. Pansinuitis right side.
7	Gross	26	M.	American	Jordan	Ozena	Friedlander	+	+	?	Neg.	Case reported greatly improved by Perez Vaccin.

8	Grove		F.	American	McLeod	Ozena	Friedlander	+	+	?	Neg.	Case reported greatly improved by Perez Vaccin.
9	Cortelyou	30	F.	American	Hopkins	Ozena	Friedlander	+	+		Neg.	
10	Belvol	7	F.	American	U. C.	Ozena	Friedlander	+	+		Neg.	Treated over year at U. C. with autog. vaccin. Unsatisfactory results.
11	Bunw	25	F.	English	Stanford	Ozena	Friedlander	+	+	16	Neg.	Clinical cure. No crusts, no odor.
12	Chun	14	F.	Chinese	Stanford	Ozena	Friedlander	+	+	13	Neg.	Clinical cure. No crusts, no odor.
13	Dougherty	14	M.	American	Stanford	Ozena	Friedlander	+	+	6	Neg.	Great improvement under treatment.
14	Devino	30	F.	Italian	Stanford	Ozena	Friedlander	+	+	13	Neg.	Great improvement under treatment.
15	Mahan	13	F.	American	Office	Ozena	Friedlander	+	+		Neg.	Great improvement under treatment.
16	Ortereras	8	M.	Mexican	Office	Ozena	Friedlander	+	+	11	Neg.	Great improvement under treatment.
17	Ortereras	7	M.	Mexican	Office	Ozena	Friedlander	+	+	7	Neg.	Great improvement under treatment.
18	Posatani	6	M.	Italian	Stanford	Ozena	Friedlander	+	+	10	Neg.	Great improvement under treatment.
19	Parenti	28	M.	Italian	Stanford	Ozena	Friedlander	+	+	9	Neg.	Great improvement under treatment.

CHART IV.

Analysis of Cases of Syphlitic Ozena.

Name	Age	Sex	Nationality	Referred by	Clinical Diagnosis	Bacteriologic Examination	Wassermann	Type of Syphilis	Odor	Crusts	1st. Ex.	Last Ex.	Result of Anti-Syp. Treat.
19954 J. B.	4	M.	American	U. C.	Syph. Ozena	Staph.	+++	Active Congenital	O	S	12-25-15	4-10-16	No change.
19161 J. W.	50	M.	American	U. C.	Syph. Ozena	Perez +	+++	Ter.	O	O	12-25-15	4-10-16	No change.
11055 A. M.	35	F.	Russian	U. C.	Syph. Ozena	Staph.	+++	Ter.	+	+		4-10-16	
11785 E. G.	37	M.	Italian	U. C.	Syph. Ozena.	Fried.	+++	Tabes.	+	+	12-25-15		
18821 A. J.	9j	F.	American	U. C.	Syph. Ozena	Perez +	+++	Active Congenital	+	+	12-25-15	4-10-16	No change.
19102 R. E.		F.	American	U. C.	Syph. Ozena	Staph.	+++		O	+	12-25-15		
19709 E. H.	37	M.	American	U. C.	Syph. Ozena	Perez +	——	Sec. Active	O	+	12-25-15	2-6-16	No change.
19348 A. G.	25	M.	Italian	U. C.	Syph. Ozena	Gram-Bac?	+++	Sec. Active	+	S	12-25-15		

CHART V.

Showing Results of Treatment with Perez Vaccine on Cases of Clinical Ozena, Friedlander and Perez Type.

No.	Name	Bac. Ex.	1st. Inj.	Last Inj.	No. of Inj.	Notes, 1st Ex.	Notes, Last Inj.	Patient's statement as to improvement in general condition	Notes
1	Miranda J.	Perez.	Apr. 10, 1915	Apr. 12, 1916	12	Sept. 22, 1914 Greenish crusts, odor offensive	April 12,1916 No crusts, no odor	General condition much improved	Stayed away one year, odor better as whole but nose must be washed from time to time. Clinically improved, but not cured. Still under treatment.
2	Miranda H.	Perez.	Apr. 13, 1914	Apr. 12, 1916	9	Apr. 13, 1914 L. nostril completedly blocked, odor very marked	April 12, 1916 No crusts, no odor	General condition much improved	Notes as with M. J.
3	Novani	Perez.	July 26, 1915	Oct. 9, 1915	12	July 26, 1915 Typ. ozena, crusts in entire nose	Oct. 9, 1915 No crusts, slight odor	General condition much improved	Pat. father does not believe in vaccines, admits great improvement.

CHART V. (Continued)

No.	Name	Bac. Ex.	First Inj.	Last Inj.	No. of Inj.	Notes 1st Exam.	Notes Last Exam.	Patient's statement as to improvement in general condition	Notes
4	Plant	Perez.	July 8, 1915	Feb. 4, 1916	26	July 8, 1915 Heavy crusts, foul sweetish odor.	Feb. 4, 1916 No crusts, no odor	Mental condition much improved. General condition vastly changed for better.	Discharged Feb. 4, 1916, clinical cure. Injections stopped for present, no odor since Jan. 11, 1916.
5	Stefanovich	Perez.	March 9, 1916	Apr. 12, 1916	11	March 9, 1916 Slight crusts, odor strong at 4 ft.	April 12, 1916 No crusts, no odor	General condition much improved.	Clinical cure, discharged.
6	Spooner	Perez.	Feb. 18, 1916	Feb. 26, 1916	5	Feb. 18, 1916 No clinical ozena in nose. Ozena of larynx, slight crusts and redness.	Feb. 26, no crusts in interarytenoid space, muc. mem. distinctly reddened and not so shiny.	Subjective improvement in larynx marked.	Discharged for 3 mos.
7	Sister P.	Perez later Fried.	Apr. 5, 1915	Nov. 15, 1915	20	Aug. 6, 1911 Pansinuitis, crusts odor, various operations.	Nov. 10, 1915 In treatment 4 years, no pus, crusts or odor.	Tremendous improvement.	Clinical and bacteriologic cure probable, repeated bac. ex. Perez neg.
8	Weber	Perez	Dec. 24, 1915	Mar. 22, 1915	13	Dec. 24, 1915 Crusts slight, strong sweetish odor.	March 22, 1915 Perez neg. since March 3, no crusts, no odor.	Tremendous improvement.	Clinical and bacteriologic cure probable.

9	Jooley	Staph.	Feb. 16, 1916	Feb. 25, 1916	7	Feb. 16, 1916 True clin. ozena, crusts and odor	Feb. 25, 1916 No crusts, no odor	Tremendous improvement.	Did not return. Probably Perez infection. Clinical cure patient's standpoint.
10	Morgan	Perez	Mar. 9, 1916	Mar. 13, 1916	4	Mar. 9, 1916 Foul char. odor. Large crusts.	Mar. 13, 1916 No odor, few crusts.		Clinical cure patient's standpoint, local improvement wonderful.
11	Posatani	Fried.	Mar. 24, 1916	Apr. 12, 1916	7	Nose filled with black crusts, odor foul at 6 ft.	April 8, 1916 Slight odor and crusts. Contact.		Improvement remarkable. Under treatment.
12	Parenti	Fried.	Feb. 19, 1916	Mar. 14, 1916	9	No crusts, slight odor.	No crusts, no odor.	Much improved.	Did not return.
13	Devino	Fried.	Feb. 24, 1916	Apr. 13, 1916	10	Crusts l. only, odor marked, not typ.	No crusts, slight odor.		Under treatment. Results unsatisfactory.
14	Dougherty	Fried.	Mar. 28, 1916	Apr. 13, 1916	3	Nose blocked, extremely offensive.	No improvement.		Under treat. Results unsatisfactory.

CHART V. (Continued)

No.	Name	Bac. Ex.	1st Inj.	Last Inj.	No. of Inj.	Notes on 1st Ex.	Notes on last Ex.	Patient's statement as to improvement in general condition	Notes
15	Adolphus	Perez. Later Fried.	Dec. 15, 1915			Dec. 1, 1915 Typ. heavy crusts, foul, overpowering sweetish odor	April 2, 1916 No crusts, no odor	Very great increase weight and appetite, mentally better	Case still under treatment, clinical not bacteriological cure.
16	Alpert	Perez	Nov. 29, 1915	Feb. 8, 1916	9	Nov. 29, 1915 No crusts, foul sweetish odor.	April 3, 1916 Nose not washed for one month, no crusts, no odor.	Same statement as above.	To date, clinical but not bacteriological cure.
17	Faber	Perez	Mar. 11, 1916	Mar. 20, 1916	6	After 11 days without wash, few crusts, medium but characteristic odor.	March 20, 1916 No crusts, no odor.	Same statement as above.	Very irregular in attendance, permanent cure doubtful, clinical cure from patient's standpoint.
18	Griffin	Perez	June, 1915	Dec. 27, 1915	25	Violent frontal headache, crusts must be removed with instruments twice a week, odor unbearable to friends. Aug. 26, 1915	No odor, no crusts, Dec. 27, 1915	Same statement as above.	Clinical but not bacteriological cure. Case refuses further treatment.

19	Hillman	Perez	Aug. 13, 1915	Jan. 27, 1916	18	Aug. 13, 1915 Crusts slight, odor not noted, patient says odor is always complained of by surrounding.	Feb. 4, 1916 No odor, no crusts.	Same statement as above.	Apparently clinically cured.
20	Kelleher	Perez				July, 1915 Crusts completely block nose, foul overpowering odor.	April 2, 1916 No crusts, no odor.	Same statement as above.	Clinical cure. Case still under treatment.
21	Luoto	Perez	Apr. 8, 1916	Apr. 12, 1916	3	April 8. No crusts, no odor.	April 12, 1916 No crusts, no odor.		Clinical cure. Case still under treatment.
22	Miranda C.	Perez	Mar. 31, 1916	Apr. 12, 1916	4	March 31, 1916 Slight crust, no odor, formerly strong.	April 12, 1916 No crusts, no odor.		Clinical cure. Case still under treatment.

CHART VI.

General Survey of Rabbit Experiments to Obtain Agglutinating Sera.
Dosage: 5 cubic centimeters of salt solution added to 24 hour growth on agar and emulsion made.

No.	First Injection	Last Injection	Total No. of Injec.	Highest Dose	Live or Dead Cultures	Date of Death	Autopsy Condition of Turbinates only	Culture	Agglutination	Culture	Notes
1	Oct 17/14.	Oct. 23/14	2	0.75 c. c.	Killed	Oct. 30/14	Accidental death Ant. turb. distinctly congested.		Negative	Original Vienna	
2	Oct. 17/14	March 15/15	11	5.0 c. c.	Killed	March 16/15	Intense engorgement ant. end inf. turb.		Negative	Original Vienna	Lived six months. Death followed overdose.
3	Oct. 23/14	Nov. 4/15	3	2.0 c. c.	Killed	Nov. 23/15	Congestion ant. turb. Pus.	Perez neg.	Negative	Original Vienna	Killed by chloroform.
4	Oct. 23/14	Dec. 23/14	5	5.0 c. c.	Live	March 10/15		Perez neg.	Negative	Original Vienna	Killed.
5	Nov. 20/14	Dec. 23/14	4	2.0	Live	Feb. 23/15	Marked atrophy of turbinates.	Perez neg.	Negative	Original Vienna.	Average loss of weight 200 gm. between injections.
6	Dec. 7/14	Dec. 23/14	3	2.0	Killed	Jan. 2/15	Congestion of turb.	Perez neg.	Negative	Original Vienna	Death due to general Perez infection.

7	Dec. 15/14	Dec. 23/14	2	2.0	Killed	Dec. 23/14	Intense congestion	Perez neg.		Original Vienna	Death from fatal dose, 12 hours.
8	Dec. 15/14	Dec. 15/14	1	2.0	Killed	Dec. 15/14	Intense congestion.	Perez +?		Original Vienna	Death from fatal dose, 12 hours.
9	Dec. 16/14	April 1/15	2	2.0	Killed	April 1/15				Original Vienna	Death from fatal dose, 14 hours.
10	Nov. 27/14	Nov. 27/14	1	1.5	Killed	Nov. 27/14	Intense congestion			Original Vienna	Death from fatal dose, 8 hours.
11	Dec. 16/14	March 15/15	3	0.75	Killed	March 15	Intense congestion.			Vienna	Death from fatal dose, 12 hours.
12	Dec. 22/15	Dec. 27/15	1	0.5	Live	Dec. 27/15				Griffin	Death from fatal dose, 4 hours.
13	Dec. 22/15	Feb. 21/16	8	1.0	Live	Mar. 11/16	Bluish black marked atrophy nose filled pus.	Perez +	Strongly positive	Original Vienna	Lived two and a half months. Death from general Perez infection.
14	Dec. 22/15	March 15/16	13	1.0	Live	Feb. 19/16			Strongly positive	Original Vienna	Lived two and a half months. Death from general Perez infection
15	Dec. 22/15		1	0.2	Live	Dec. 22/15				Plant	Fatal dose. Death in 4 hours. Baby rabbit, weight 230 grams.
16	Dec. 22/15		1	0.2	Live	Dec. 25/15				Strum	Fatal dose. Death in 4 hours. Baby rabbit, weight 250 grams.

CHART VI.

General Survey of Rabbit Experiments to Obtain Agglutinating Sera.
Dosage: 5 cubic centimeters of salt solution added to 24 hour growth on agar and emulsion made

No	First Injection	Last Injection	Total No. of Injec.	Highest Dose	Live or Dead Cultures	Date of Death	Autopsy Condition of Turbinates only	Culture	Agglutination	Culture	Notes
17	Dec. 22/15	Jan. 21/16	7	2.3	Live	Feb. 4/16	Marked atrophy	Pure Perez	Strongly positive	Adolphus	Cause of death unknown. Apparently in good condition.
18	Jan. 21/16		1	0.4	Live	Jan. 23/16	No change.			Alpert	Cause of death unknown.
19	Dec. 27/15		1	0.2	Live	Dec. 27/16	Acute conges.			Femol	Fatal dose. Died 4 hours. Baby 230 grams.
20	Jan. 7/16	Jan. 10/16	2	0.6	Live	Jan. 13/16	Slight conges.	Staph.		Griffin	Fatal dose. Baby, 2½ months.
21	Jan. 7/16	Jan. 27/16	4	0.8	Live	Feb. 12/16		P. C. Perez	Negative	plant	Baby, 2½ months. Lived 3 months.
22	June 15/15		1	5.0	Live	Dec. 6/15		P. C. Perez	Positive	Vienna	Survived one enormous dose of 24 hour agar growth.
23	Jan. 7/16	Jan. 10/16	2	0.6	Live	Jan. 17/16	Destruction of turb.	P. C. Perez	Positive	Femol	Baby, 2½ months. Greatly emaciated at death.

24	Dec. 22/15	Dec. 31/15	8	1.0	Live	Feb. 7/16			Positive	Kelleher	Doses given at four day intervals.
25	Dec. 27/15		1	0.2	Live	Dec. 30/15		P. C. Perez	Positive	Vienna	Baby, 230 grams. Acute coryza following inj.
26	Jan. 10/16		Inoculation of nose		Live			P. C. Perez	Positive	Vienna	Baby, 2½ months. Exp. for direct transference. Still living.
27	Jan. 10/16		Inoculation of nose		Live			Perez negative		Vienna	Baby, 2½ months. Exp. for direct transference. Still living.
28	Dec. 10/15		1	0.2	Live	Dec. 11/15				Vienna	Baby, 244 grams. Ex. to det. size of fatal dose.
29	Dec. 31/15		1	0.5	Live	Dec. 31/15	Intense congestion.			Plant	Died in 4 hours; fatal dose.
30	Dec. 31/15		1	0.4	Live	Dec. 31/15				Jackson	Died in 4 hours; fatal dose.
31	Dec. 31/15		1	0.5	Live	Dec. 31/15	Intense congestion.			Griffin	Died in 4 hours; fatal dose.
32	Dec. 10/15		1	0.5	Live	Dec. 10/15				Vienna	Died in 7 hours; fatal dose.
33	Dec. 31/15	Feb. 9/16	8	1.0	Live	Feb. 21/16			Positive	Femol	Baby, 1st dose 0.1 c.c. increased by 0.1 each dose. Last dose fatal.

CHART VI.

General Survey of Rabbit Experiments to Obtain Agglutinating Sera.
Dosage: 5 cubic centimeters of salt solution added to 24 hour growth on agar and emulsion made

No.	First Injection	Last Injection	Total No. of Injec.	Highest Dose	Live or Dead Cultures	Date of Death	Autopsy Condition of Turbinates only	Culture	Agglutination	Culture	Notes
34	Dec. 7/15	Dec. 7/16	3	0.6	Live	Dec. 7/16		P. C. Perez	Positive	Mixed	Strong agg. serum after 2nd inj. Wt. 1340 grams.
35	Dec. 3/15	Jan. 21/16	3	0.3	Live	Jan. 21/16	Slight congestion	P. C. Perez	Positive	Vienna	Baby rabbit.
36	Nov. 29/15	Dec. 23/15	3	2.0	Killed	Dec. 20/15	Slight congestion	P. C. Perez	Positive	Alpert	Lost 200 grams after two inj.
37	Nov. 29/15	Dec. 13/15	3	1.0	Killed	Dec. 13/15				Griffin	Fatal dose.
38	Dec. 13/15	Dec. 17/15	2	0.4	Live	Dec. 25/15	Atrophy and pus pockets	P. C. Perez	Positive	Vienna	Strong agglutination, even with two injections. Reduced to skeleton.
39	Jan. 27/16	Mar. 29/16	8	1.4	Live	April 14/16	Atrophy and pus.	P. C. Perez	Positive	Vienna	Killed.
40	Jan. 27/16	Feb. 14/16	5	2.0	Live	Feb. 25/16	Atrophy and pus.		Positive	Vienna	Killed.
41	Jan. 27/16	Feb. 14/16	5	2.0	Live	Feb. 25/16			Positive	Alpert	Killed and bled. Strong agglutination.

42	Jan. 27/16	Feb. 14/16	5	1.8	Friedlander.				Negative	Eugene O.	No agglutination for any Fried. strains. Rabbit living and well.
43	Jan. 31/16	Mar. 15/16	7	2.0	Perez, Live				Positive	Jackson	Agglutination strong after fourth injection.
44	Feb. 9/16	Feb. 25/16	4	1.5	Perez, Live					Piffle	Killed and bled. Agglutination weak.
45	Feb. 9/16	Mar. 29/16	7	2.0	Perez, Live	April 24/16				Kelleher	
46	Feb. 14/16	Mar. 15/16	6	2.0	Perez, Live					Jackson	Rabbit living.
47	Feb. 21/16	Mar. 3/16	4	2.0	Perez, Live	Mar. 12/16				Plant	Died; no autopsy.
48	Feb. 21/16	Mar. 29/16	6	2.0	Perez, Live					Adolphus	Rabbit living.
49	Feb. 21/16		1	0.5	Mixed Live	Feb. 22/16				Kenealy	Friedlander and Streptococcus.

XI.

EXPERIENCE WITH SUSPENSION LARYNGOSCOPY IN OVER TWO HUNDRED CASES, WITH REPORT.*

By Joseph C. Beck, M. D.,

Chicago.

To criticise fairly any new method, one should have sufficient experience in order neither to be overenthusiastic and so mislead men into abandoning the well-tried and successful methods, nor to report discouragingly and so engender at the outset prejudice against giving a new procedure a trial. Having previously made this same mistake, and being aware of others who have done likewise, I have purposely avoided the publication of our results with suspension laryngoscopy until this day, after three years' experience in more than two hundred cases in private practice.

We are again indebted to Killian for a great contribution to science and to laryngology in particular; and I wish to say, at the outset, that I believe suspension laryngoscopy is destined to become one of the greatest helps in the diagnosis and surgical management of diseases of the larynx, trachea, bronchi and esophagus.

The evolution of the method from its first accidental discovery while Killian was working on a cadaver, up to the present time, when Lynch has perfected the instrumentarium, is most interesting and can be found in the new edition of Jackson's textbook on Peroral Endoscopy and Laryngeal Surgery.

It is to Freudenthal that I acknowledge my first clear information on this subject, when he demonstrated the method before the American Laryngological, Rhinological and Otological Society, three years ago. With the first type of Killian's instruments (Figure 1) I began experimenting in the examination of various cases, and found it most difficult and unsatisfactory. I was only able to succeed with general anesthesia, and even then not with absolute satisfaction. One afternoon, while demonstrating the method to Dr. Lynch, who was visiting me, I noted his criticism and enthusiasm, and in less than six months I found, to my great satisfaction, that most of my first objections were overcome by his perfection of instruments and technic, as well as by his addition of many new features, which he demonstrated by treating a number of patients with particularly good results.

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*Read before the Chicago Otolaryngological Society, November 16. 1915.

During the progress of Lynch's work, a number of European articles appeared as well as newer instruments, particularly those of Albrecht, and Bruning's counter pressure attachment (Figure 2), which I found to be an improvement on Killian's original (Figure 1), as well as on Killian's improved instrument (Figure 3).

Until I obtained the last model of Lynch's instrument (Figure 4), the greatest difficulties with us have always been: (1) To prevent the tongue from sliding to one side or the other; (2) to keep the epiglottis on the point of the spatula; and (3) our inability to expose the anterior commissure in every case. I also noted that following the use of the apparatus the patient complained considerably of soreness, or at least of an uncomfortable feeling at the base of the tongue and in the teeth.

Several patients I was unable to suspend on account of certain pathologicoanatomic conditions, such as: (1) The presence of long upper central incisors, there being no other teeth present in the upper jaw; (2) a stiff neck from an old cured scoliosis, preventing extension of the neck; (3) a tumor at the base of the tongue in the region of the valecula; (4) complete absence of the teeth in the upper jaw, with a great deal of absorption of the alveolar process; (5) very loose teeth in patients with pyorrhea alveolaris; and (6) partial ankylosis of the lower jaw. In addition to these there were some individuals who, in spite of the most thorough local anesthesia, would continue to react and thus foil any attempt at suspension. These were probably due to mental anxiety.

Some of the conditions in which we have employed suspension laryngoscopy are: (1) Carcinoma, (2) adenocarcinoma, (3) papilloma, (4) fibroma, (5) angioma, (6) syphilis,

(7) tuberculosis, (8) rhinoscleroma, (9) cicatricial stenosis, (10) paralysis, (11)) foreign bodies, (12) burns, (13) asthma, (14) posttraumatic tracheal stenosis, (15) bronchiectasis, (16) suction of secretion for bacteriologic examination, (17) direct intubation, (18) simple laryngitis, (19) posttyphoidal chondritis, (20) atrophic laryngotracheitis, (21) hysteric aphonia, (22) suspected thymus enlargement, (23) tracheal compression by thyroid gland or tumor, (24) aortic aneurism, (25) normal cases.

INSTRUMENTS AND TECHNIC.

Instruments.—(1) Table with the Lynch extension (Figure 5), (2) crane, (3) hook, (4) spatulæ, (5) illumination (Beck's lamp) (Figure 6).

Technic.—(1) Anesthesia. This may be local by applying cocain twenty per cent and injecting novocain one per cent into superior laryngeal nerve; or general by the use of ether vapor.

(2) Position of patient. Patient is in recumbent position, with head in extension over edge of table.

(3) Introduction of instrument as far as epiglottis, then under it—i. e., on its ventricular surface.

(4) Counter fixation on the upper incisors by the tooth plates.

(5) Opening of mouth to a space sufficient for working purposes.

(6) Hook is placed on the transverse bar of the crane, while patient's head is kept well in the median line.

(7) Horizontal bar is raised by turning the vertical portion of the crane, thus lifting head off the table.

(8) The larynx and hyoid bone are brought forward and further upward by turning the handle of the horizontal portion so that the latter moves toward the operator.

(9) The larynx is fixed and the anterior commissure brought more into view by pressing the thyroid cartilage down with the left hand, in preference to the Bruning's counter pressure instrument.

(10) The right hand is left free for any manipulations in the matter of treatment, diagnosis, operations or further manipulation of the crane. For the removal of tumor, either for diagnostic or operative purposes, we have found that the

Cordes instrument with a set of five tips (Figure 7) has served us the best. One should always have on hand a substitute, and one of the best is the Killian short alligator bronchoscopic forceps (Figure 8). A long handled ring curette (Figure 9) and a long dressing forceps for sponging or removing particles of tissue (Figure 10) make up the instrumentarium. The elaborate and well devised set of instruments of Dr. Lynch are unquestionably of value, especially in the hands of such an expert as himself; but we have been able to manage with those mentioned.

We have never sutured any part of the larynx from within under suspension, but we have made use of the actual cautery in several cases of ulceration, especially of the tuberculous type, and we found a long electrode with especially heavy blade of greatest service (Figure 11). The suction apparatus is of great assistance in the work, and an atomizer with cocain and adrenalin should always be on hand, in order to supplement the anesthetic while working with the local anesthesia. Morphin and atropin are excellent accessories to good work.

When general anesthesia is required, it is well to employ the vapor method; and ether is the drug of choice, although we have used nitrous oxid and oxygen in one case, and ether and oil per rectum, also scopolamin—morphin in several other cases. In a number of cases in which tracheotomy had been performed previously or at the time of the suspension procedure, the intratracheal method of administering ether or chloroform was employed.

CASE REPORTS.

Case 1.—Mr. M., forty-six years of age, was referred to me by a laryngologist, with the diagnosis of adenocarcinoma. The doctor had removed a piece of tissue for microscopic examination, and this, with the pathologist's report, was furnished by the patient. Symptoms were hoarseness and some embarrassment in breathing. General condition was good, although the patient had lost some weight.

Examination under suspension laryngoscopy revealed a small growth, irregular in outline, situated on the left vocal cord, near the anterior commissure. Arytenoids moved freely, and there was no enlargement of cervical glands. A piece of tissue was removed, and all the physical and laboratory tests, including the Abderhalden, were made. Wassermann was negative, Abderhalden positive; physical examination was negative. Microscopic diagnosis of the pieces of tissue removed was adenocarcinoma.

Treatment.—Twelve deep X-ray treatments, by means of the Coolidge tube, were given over different areas of the larynx within two weeks. Then by means of suspension laryngoscopy and the Cordes biting forceps (Figure 7), all the growth was removed. Two days later the deep X-ray treatments were again resumed. About one week later the patient exhibited symptoms suggestive of hyperthyroidism, and he was given thyroid extract tablets, which he has been taking for four months according to indications.

After these procedures, reexamination of the larynx showed a complete disappearance of the adenocarcinomatous growth. Patient's voice was clear, and breathing was normal. At this time he went East for a two months' vacation, so I advised him to consult Dr. Edgar Holmes, from whom I received the following report:

"It was a pleasure to see your laryngeal malignant patient, especially as the present results seem so brilliant. I sincerely hope, for his sake, that there will be no recurrence. His phonation seems to cause so slight strain and little clanging of cords, that his chances seem much better than usual."

When the patient returned to see me again I found that there was no recurrence, and after two more weeks under observation he left in excellent condition.

The following telegram was just received from the patient:

"Nanaimo, November 13, 1915.

"Boucher away at war. Dr. Wilkes, who was treating me with Boucher, says no sign of growth. Good movement; practically no swelling or congestion; excellent voice; throat could hardly be better. Feel very well; weight about same as when in Chicago. Writing tonight. C. R. MASTER."

Case 2.—Master Kty., a boy, seven years old, who had been hoarse since infancy and recently complained of difficulty in breathing.

Examination by means of the laryngeal mirror, which was very easily performed, revealed a typical papillomatous mass filling the subglottic space, including the cords. An operation was decided upon and performed. Suspension was employed, but as it was impossible to perform it under local anesthesia a general anesthetic was administered. As considerable difficulty in breathing resulted, it was decided to do a tracheotomy, which would also facilitate the healing and operation. While doing the tracheotomy the boy vomited a large quantity of thick material consisting of potatoes, etc., and we were fortunate in having at hand the suction apparatus to prevent inspiration of the vomitus. As soon as the tracheotomy was made, he was given the vapor ether anesthesia by that route. He was then very readily suspended and the growth removed. It was shown subsequently to be a papilloma by microscopic examination. Patient was put to bed in good condition. He had a fairly good night, but in the afternoon of the next day it was noted that his respiration increased to forty and his temperature to 101.6° with some cough. He complained of some pain in the left precordial and upper left abdominal region. It was also noted that he had practically no pulse on the left side, whereas on the right radial side his pulse was very distinctly felt. A probable diagnosis of bronchopneumonia was made by Dr. Meyers, and patient was placed under suitable treatment for such condition. The next day, the third after the operation, I saw him and found practically the same condition as described above, and in consulting with Dr. Friedberg decided that he probably had a pneumonia, due, I thought, to inspiration of some food particles, but Dr. Friedberg attributed it to the anesthetic. That evening when I left the hospital he had rallied some. A special nurse was put on the case, and she telephoned me about nine o'clock that his temperature had gone down to a little over one hundred, and he appeared to breathe easier. About ten-thirty o'clock he asked her for some drinking water, and the next moment she noticed a sudden gush of blood from the tube and the mouth. The quantity of blood was considerable. He immediately sank and died in a few moments, in spite of heroic efforts to save the child. Postmortem examination was denied. It is a question why he had a pulse only on the right side and not on the left. It is not likely that it was an anomalous course of the radial, because he had been in the hospital for a few days before, and this no doubt would have been discovered; besides, I inquired of the family physician who had taken care of the boy, and he never discovered this.

Case 3.—Mrs. M., who has been hoarse for nearly two years and under the care of Dr. Denman of Toledo, a laryngologist, who had been treating her for a growth on the vocal cord. This growth has been there for nearly two years. It was operated upon by direct laryngoscopy by Dr. Meyers of Ann Arbor, and a small piece removed, which was proven by the microscope to be a granuloma. It appeared to return very soon, and the patient then consulted Chevalier Jackson, who declared it to be most probably a fibroma, and had the patient desist from loud speaking for several months. While this treatment did not cause the growth to disappear, nor the hoarseness to improve, Dr. Denman did note that the growth, which was always of a cherry red color, became paler when patient employed whispering voice.

When I saw the case last July in Toledo I found a small, bright red colored neoplasm on the left vocal cord, near the anterior portion. It was decided to remove this growth, but the patient would not consent to having it done under local anesthesia, since she seemed to have had considerable pain during the previous manipulations. Dr. Denman, who employs nitrous oxid and oxygen anesthesia for most of his work, and has an expert to give it, urged me to try to remove this growth under suspension and gas anesthesia. I consented, and to our great surprise it was not at all difficult. The patient awoke immediately after the growth was removed and larynx inspected to see that all was well. She did not, however, utter a sound. I was sure that this was probably only transitory, because we had the same thing occur before. After about two weeks I received a letter from the doctor, stating that aphonia was no better, although the cords were smooth and their movements normal. Two months later the doctor brought the patient up because the voice had not returned, and since the voice was of a potential value to the patient, considerable anxiety was felt by all. I found that the cords looked good and their movements were normal, but the patient could not speak above a whisper. Still expressing my belief to the patient that she would speak, and advising the doctor to continue expectant treatment, the patient went home somewhat more satisfied. Yesterday I received the following telegram, in answer to my inquiry:

"Toledo, Ohio, November 10, 1915.

"Dr. Joseph C. Beck: Cord smooth, straight, slightly congested; phonation most words possible, but difficult and fatiguing; quite hoarse; steady slow improvement.

"IRA O. DENMAN."

I have found no reference to this complication, but I believe that it is functional (hysteria) and not organic, since it occurred so rarely. It will, however, bear remembering and possibly study from another standpoint—namely, traction trauma, with possible muscular relaxation. The microscopic examination showed angiofibroma.

Case 4.—Mr. W., forty-two years of age. A very large healthy looking man. Hoarse for about six months. A laryngologist of his city, who referred him to me, stated that he observed the condition change frequently, whether under treatment or not. Examination showed the right arytenoid fixed and an irregular appearing growth on that side and somewhat subglottic, also involving part of the cord. By means of suspension a particle of the growth was removed, and the microscope showed it to be sarcomatous; yet the Abderhalden test for carcinoma was positive, while the Wassermann was negative.

Following the removal of the growth there resulted considerably more bleeding than normal, which continued for some time; so while the patient was suspended we introduced a large intubation tube, which controlled the hemorrhage beautifully.

EXHIBITION OF LARYNGEAL CASES, WITH SPECIAL REFERENCE TO SUSPENSION LARYNGOSCOPY.

The first case presented is a patient whose condition was diagnosticated as scleroderma by men who are supposed to be authorities in dermatology. Scleroderma is a condition in which there is this deformity in both hands, and probably also of the eye (Figure 12). Prof. Shamberg of Philadelphia has also found evidences in the larynx. In the case presented, the patient had a marked laryngeal obstruction that required tracheotomy, and subsequently laryngostomy was necessary. The cartilage was unusually hard, and there was difficulty in cutting through it. We were required to use bone forceps for this purpose. There is, as will be noticed when the man swallows, complete separation of the thyroid cartilage now. There is an up and down tube placed into this slit (Figure 13), through which he breathes, one part going into the mouth, the other into the trachea. It is interesting that the condition of scleroderma occurs on the mucous membrane of the larynx, producing an irreparable condition and requiring such procedures as that performed in this case. Examination of the larynx from within simply shows the smooth surface of the vocal cords, with immovable arytenoids. There is still a plastic operation to be performed in this case after dilatation has been accomplished.

In splitting the larynx, after some few days, we noticed a white area, which we thought was caused by the pressure of the tube. For this reason we left the tube out for a few days, but found that this white, thick area on each side of the cricoid was still present. We excised it, and microscopic examination showed a hyperplasia of the mucous membrane and scar tissue. The case is too recent for any definite conclusions regarding it.

The second case is that of a man who came to the Cook County Hospital several years ago, requiring an emergency tracheotomy. He was fifty-six years of age, and had been hoarse for over three years. Although carcinoma was suspected in the case, he gave a very marked four plus Wassermann. He was given antisyphilitics, and subsequently a laryngostomy was performed. In closing the wound the plastic method was employed, using the skin. Two lateral incisions were made, the skin turned in and sutured in the median line. This patient has a defect in the thyroid cartilage, which can easily be felt. He is a laborer, and gets along very nicely, although there is still considerable difficulty in breathing when he has to exert himself. This case which is cured, is presented because of an interesting finding recently. In textbooks on dermatology and plastic surgery, it is said that whenever you transplant an area with hair into an area or region where there is mucous membrane, or where hair does not exist, the hair follicles die in the transplant. Nine months after the plastic in this case the man returned, complaining of a tickling in his throat. In the writer's absence, Dr. Pollock looked in his throat and found what he took to be a black thread there. The writer then suspended the patient and found that this black thread was a bunch of black hair, which could be pulled out easily. Microscopic examination substantiates the presence of hair. To devise a method to destroy these hair follicles, if this hair returns, will be the next thing, and the X-ray is probably the only thing to be thought of at the present time. The man is gray haired, and yet the new hair is black. (Figures 14 and 15.)

The next patient is presented principally because it was impossible to suspend her, on account of the existence of a tumor at the base of the tongue. A woman, about thirty-five years of age, has had for several years the sensation as though there was something on the back of the tongue. In the past few months she had some difficulty in breathing and swallowing, as well as a change in her voice. These symptoms all increased in severity, so that the family physician referred her to a throat specialist, who in turn consulted me. Examination revealed a rounded looking tumor, smooth in character, about the size and shape of a half of a walnut and somewhat elastic but not painful on phonation. It did not involve the epiglottis, but pushed it backward so that it interfered with inspiration with a sort of valve action. I made a tentative diagnosis of thyroglossal duct cyst, and recommended puncture for diagnosis. We withdrew a very small quantity of a rose colored fluid, which the microscope showed contained a few red blood corpuscles; cultures remained sterile. We then decided that it was probably an accessory or internal thyroid gland, of which I have had one case, and decided upon operation. We believed that we could do so by the suspension method. On attempting to do so, however, we found it impossible, since the spatula could not be placed far enough back. I decided at that time to devise a spatula that would have a space in the center of its terminal or epiglottic end to permit the tumor to fit in and the side prongs of the spatula depressing the base of the tongue—a sort of a two-pronged fork—and thus enable one to employ suspension in similar cases. We did not wait for the construction of this spatula, however, but operated in the following manner, with excellent final results:

Patient was too irritable to do the operation under local anesthesia, so ether was employed. As soon as patient was anesthetized, it was found impossible to keep the tongue and, consequently, the tumor from closing off the larynx, so it became necessary to do a hurried tracheotomy through a very short fat neck. Subsequent anesthesia was administered through this tracheal canula by the vapor method. It was soon discovered that the removal of the growth would be very difficult, if at all possible, by the oral route, so we determined to do an external pharyngotomy, which was comparatively easy of execution. The suction apparatus was particularly valuable for the removal of the blood and secretions of the pharynx. The tumor, which was very easily liberated from the tongue tissue proper, appeared to have a capsule. Its actual size is seen in Figure 16, and the macroscopic examination on cross section suggested a colloidal degenerated thyroid gland. Subsequent microscopic examination proved it to be such. The bleeding was easily controlled by a hypopharyngeal packing for twenty-four hours. The patient made an uneventful recovery, having some difficulty in swallowing, at first. She was kept on rectal feeding for five days, the tracheal tube was removed at the end of a week, and now one can see a smooth scar at the site of the previously located tumor. All the normal functions are preserved.

The next case is presented for many reasons of interest to the laryngologist. He was shown to you earlier in the evening when I showed the automatic closing of the tracheotomy tube. This man, who is about thirty years old, a machinist by trade, was first seen by me two years ago, when I found him suffering with chronic laryngeal obstruction of some sort, demanding urgent action to give him air. An emergency tracheotomy was performed and a correct diagnosis sought. At that period I left my service, and the patient came under the service of Dr. Stanton Friedberg. On my return, three months later, I found the patient in excellent general health. Dr. Friedberg informed me that he found some pus about the arytenoid region, and believed it to be some form of chondritis. He also stated that he made several punctures and got some pus. The most thorough and painstaking examination, as well as systematic treatment, excluded syphilis, tuberculosis, and laryngeal scleroma. We therefore decided on a laryngostomy, which was performed a little over one year ago. Under general vapor ether anesthesia, the thyroid cartilage was split. We found that the arytenoids were immobile, the vocal cords transformed into marked hypertrophic masses. This same type of thickened mucous membrane extended down through the larynx and trachea, almost within an inch of the sternal notch, and so narrowed these passages as practically not to permit the introduction of a bougie one-eighth of an inch in diameter. The trachea, therefore, was also split down to the limits of this constriction. An up and down laryngostomy tube was inserted, and the larynx and trachea incisions kept apart by packing. Daily changing of the tube and redressing of the cleft for about three weeks showed that this above mentioned hypertrophic mucous membrane did not seem to disappear—on the contrary, it became thicker. We then decided to remove part of it, anticipating healing by a cicatricially modified epithelial lining. Under local infiltration anesthesia a portion was removed with considerable difficulty, on account of the extreme hypersensitiveness of the area; in fact, this was evident at each dressing, in spite of the very free use of a strong cocain solution. The up and down tube was again introduced and the laryngotracheal cleft kept apart for several weeks. During this time it developed, from the examination of his nose, that he had a suppurative process on his left side, and from the history it appeared that this condition had existed for several years. This finding led me to the conclusion that the condition of his larynx and trachea was secondary to this chronic suppurative sinuitis. The microscopic examination of the tissue removed showed it to be a true hyperplasia of the epithelium as well as of the glandular structures. Such pathologic changes are frequently reported as resulting from suppurative conditions of the nasal accessory sinuses and nasopharynx. I then attempted to do an intranasal operation on his middle turbinate and ethmoid cells, but was very unsuccessful, owing to my inability to obtain sufficient local anesthesia. After keeping the laryngotracheal cleft open for four and one-half months and dilatating the laryngeal chink, we had a very satisfactory result, and he was ready for the plastic closure or laryngostomy (Figure 17). The gap being of considerable width and length, and inasmuch as about one-half of it was of the trachea, I feared that skin flaps from the neighborhood would not suffice, in view of the danger of being sucked in with any effort at inspiration. Consequently, I sought a method of employing some solid organic material, and determined to transplant a section of a rib. It is well known that bone transplants do not do well and become absorbed in locations where there is not normally bony structure; yet I was hopeful that sufficient osteal or periosteal tissue would remain to form a firm closure. Therefore, under general nitrous oxid oxygen anesthesia by the intratracheal method, aided by scopolamin-morphin, a section of the right eighth rib, two inches long, was removed with its periosteum and put into a subcutaneous tunnel previously prepared in the side of the neck. This healed in fairly well, in spite of some suppuration (Figure 18). After three more weeks, under the same type of general anesthesia, this rib transplant with its overlying skin was again made into a flap with its hinged pedicle on the side of the laryngotracheal cleft. This brought the skin within the interior of the larynx and trachea. The opposite lateral margin of the cleft was now freshened and the flap containing the rib transplant sutured in, thus closing the cleft of the larynx and trachea. The top surface of the flap now was made up of a thick mass of granulations which should have been immediately covered with some sort of skin flap or graft. As a consequence, the granulation shrunk, leaving the bone graft without sufficient nutrition, and this procedure proved to be a failure. Two weeks later I attempted the double transfer method of grafting, by what is known as the "Kausch method"—namely, employing the second toe of left foot, engrafted into palm of right hand and subsequently transferring it into the laryngotracheal cleft. A toe was employed instead of the finger, because he is already handicapped by having lost two of his fingers by accident while at work as a machinist.

While the toe and hand were joined and sutured, they were kept immobilized by encasing the greater portion of the body in a cast. It is best to have the patient practice this position by strapping him in a jacket made of heavy buckram (Figure 19). One must see that the normal functions of the bowels and bladder are possible while in this restrained cast. All went well, although the patient was very uncomfortable, until the ninth day, when, during the night, the patient had a nightmare, and with a violent effort loosened the attachment of hand and foot. When I took off the bandage I found the union to be slight, and since the patient appeared to be much depressed by the failure, I desisted from reattaching the toe and hand and reapplying the cast. This, then, was the second failure. While discussing the case with a general surgeon (Dr.

W.) and planning to employ fascia lata transplant, he suggested that I try a flap containing a part of the clavicle.

One month from the last attempt I operated in the following manner, under scopolamin-morphin and ether anesthesia: A skin flap was made, the pedicle being located in the side of the neck and the periphery over the inner one of the clavicle. Dissecting this later portion as far as the upper and lower border of the clavicle with a large flat chisel, I removed a flat piece of this bone the entire width of the clavicle and one and one-fourth inches long. I now turned the dermal layer of the flap under this bone chip, surrounding it, so to speak, and left it in situ, so as to have it well nourished for the next step. Ten days later I loosened up the entire flap, including the bone, and turned it into the defect so that the dermal layer looked inside of the larynx. Along the side margins of the cleft, the flap was denuded of the epithelium and stitched by a few interrupted sutures into them. The primary defect over the clavicle was immediately sutured. The union of the implant was very satisfactory, and on the eighth day the pedicle was severed and the closure of the cleft perfected, as well as the remaining defect over the clavicle closed. The ultimate result from this last procedure was very satisfactory (Figure 20).

Following all these procedures, the patient left the hospital with a very good breathing tube and a very fair voice. Laryngeal examination revealed almost complete fixation of the arytenoids and a single nodular excrescence above what appeared to be the remains of the vocal cords. There was but slight vibration observed from them. The man now obtained a position as chauffeur and had practically no trouble, except whenever he exerted himself he would experience some difficulty in breathing. I decided upon dilatation by bougies, etc., by means of suspension, but found that I was absolutely unable to do this. This, I believe, was due to the laryngostomy operation, in that the newly made larynx was placed so much more anteriorly that it could not be brought into view, at least not by means of the Albrecht or Killian instruments. Yet I felt sure that I could do so by means of the new Lynch apparatus. I decided to wait for it, and on October 12th I suspended him, and with the aid of the longest spatula, I introduced Ingalls' long flexible tube, of which I took an X-ray while in situ (Figure 21). This tube was permitted to remain for three and one-half hours, and following its removal the patient declared that he breathed very freely and his voice was better than it had been for a long time. However, within an hour or two he showed signs of edema and extension of inflammation into the bronchi. He appeared to be in imminent danger of pneumonia.

On the fourth day from the time of the above mentioned procedure he breathed so laboriously that I was forced to do a tracheotomy. This was very easily performed in the upper extreme of the tracheal defect, just on the side of the clavicle graft. I found the bone still intact and the trachea and larynx smooth within, and no evidence of any constriction. The suppuration from his nasal accessory sinuses still annoys him considerably, and believing that it was keeping up the laryngeal trouble, I felt that it was advisable to operate thoroughly and to forcibly dilate the larynx, as well as remove the excrescence mentioned above.

On November 8th, three weeks from the time of performing the tracheotomy, I placed him under a general ether vapor anesthesia and suspended and dilated, with considerable ease. I also cleansed out the ethmoid and opened the rest of the nasal accessory sinuses. The suspension and extreme effort to get the much displaced larynx into view caused the spatula to come too forcibly in contact with the lower jaw, and a very disagreeable accident occurred, causing the three central teeth to turn outward and hang merely on the gingival mucous membrane. I replaced them immediately in their sockets and wired them to the neighboring teeth. He recovered from these last procedures, and now is in splendid physical condition, with respiration and voice improved. The nasal accessory sinus suppuration has practically disappeared. He is now wearing this automatic closing tracheotomy tube, so that I may be certain that the space for breathing between the vocal cords is permanently large enough.

On May 10, 1916, I closed the tracheal opening, and the patient is now breathing freely in the natural way and he has a fairly good voice.

In this connection I desire to present this new device which I call an automatic closing tracheotomy tube.

The indication for the use of this instrument is when the patient desires to speak and objects to placing his finger continually over the opening of the tube, which is conducive to irritation and infection of the wound, and which is always associated with considerable embarrassment.

Particularly is this of value in the treatment of such conditions as bilateral abductor paralysis, or irremovable obstructions below the tracheotomy tube, as from cicatrix, malignant disease, etc.

The instrument consists of a regular tracheotomy tube, the inner tube being very short, having attached to it a shutter which is operated by a flexible rod passing through the flexible cable tube. At the distal end of the apparatus is seen a spring push button, which is attached to the rod, and by compression closes the opening of the tracheotomy tube, the opening of which is again reestablished when the button recoils by spring action (Figure 22).

The entire instrument is hidden from external view, as shown (Figure 23), the button being carried through an opening into the man's trouser pocket, or in the pocket of a woman's skirt. It may pass through the sleeve of the patient, being held hidden in the palm of the hand.

Figures 24 and 25 show the instrument in action.

NOTE.—Since reading this paper I have had considerable additional personal experience and have discussed suspension laryngoscopy with many others, and have watched not a few at work. I have received the impression from all these sources that there is a greater limitation to suspension than was formerly believed. I am, however, even more firmly convinced than heretofore of its great value in the diagnosis and surgical treatment of the larynx and trachea. General anesthesia (nitrous oxid ether vapor) is of great assistance, but local anesthesia is more practical. I have had better success since I have followed the suggestion of Israel of Houston, who recommends the application of pure flaked cocain over the tongue, pharynx and larynx, thereby securing a better anesthesia than by swabbing solutions or injecting the superior laryngeal nerve.

Another valuable method which I desire to place on record is the socalled coagulation method of Percy. A copper rod heated by electricity is applied, the surrounding tissues being protected by ice cooled speculum, etc. Figure 26 shows a modified Lynch speculum in the form of a sheath, into which fits the cooling speculum or tube. The cooling process is accomplished by permitting ice water to flow through its double wall. In addition, there is passed a metal flexible tube containing two smaller tubes through which cold water flows into the upper part of the esophagus, preventing scorching of that organ. In addition, two plates resembling Leiter's coils are placed on the sides of the larynx to prevent scorching of the neck tissues. I am also using Lynch's modeling compound aluminum plates, which do away with some of the difficulties mentioned in the paper.

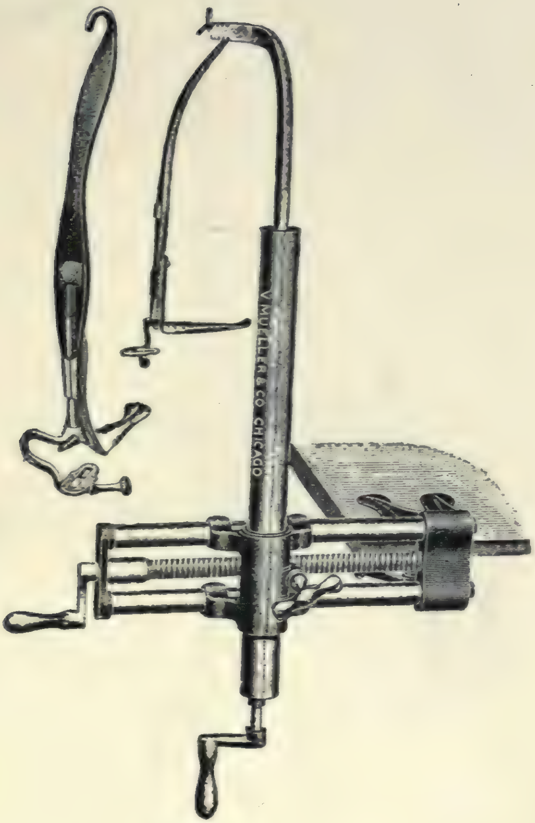

FIGURE 1.

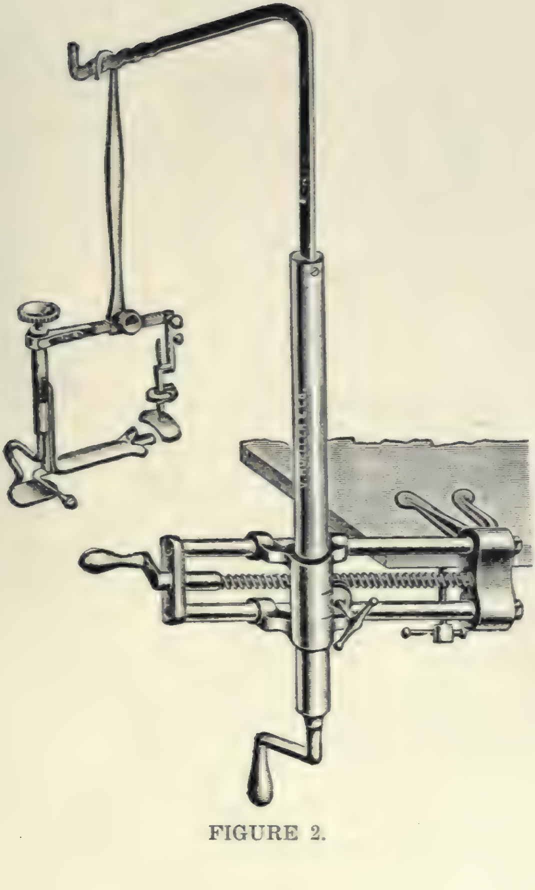

FIGURE 2.

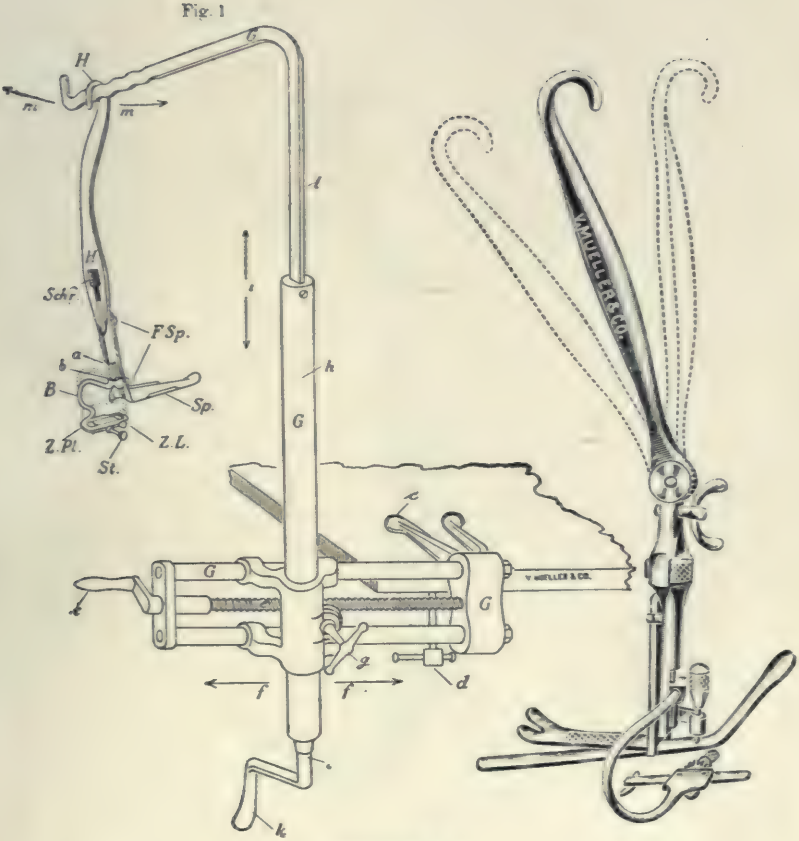
Fig. 1
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FIGURE 3.

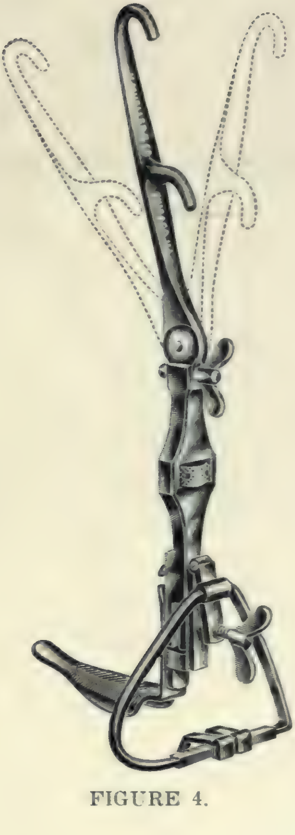

FIGURE 4.

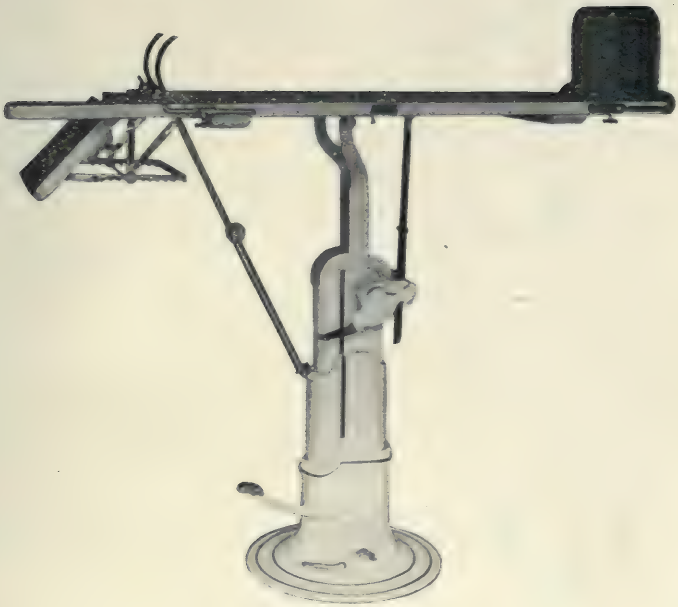

FIGURE 5.

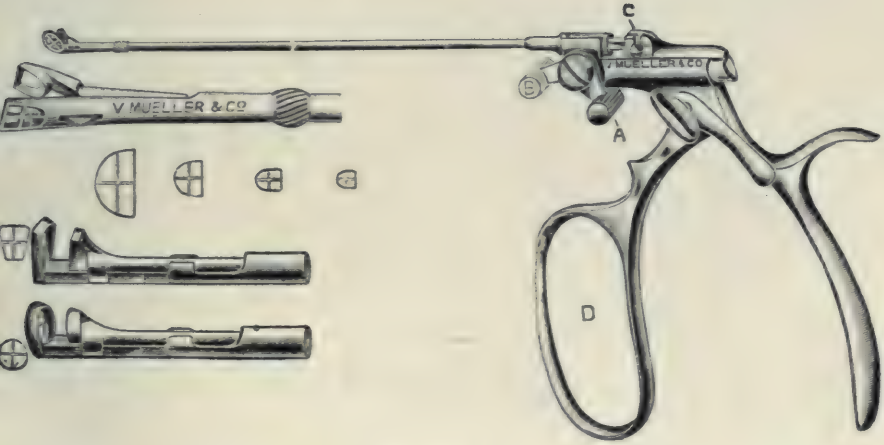

FIGURE 7.

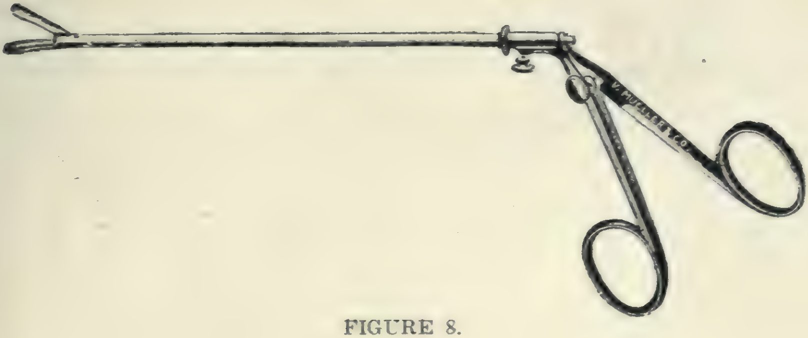

FIGURE 8.

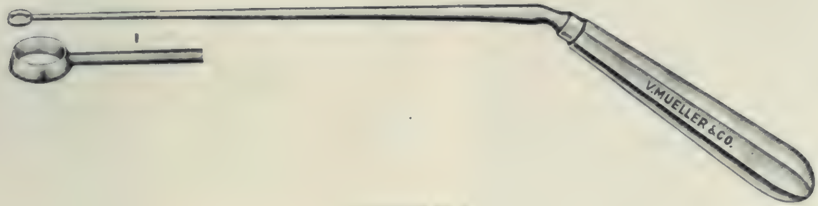

FIGURE 9.

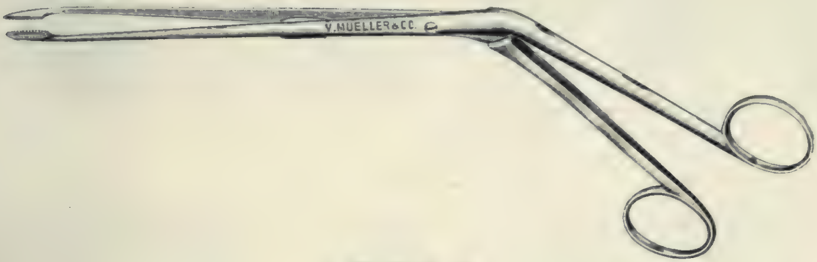

FIGURE 10.

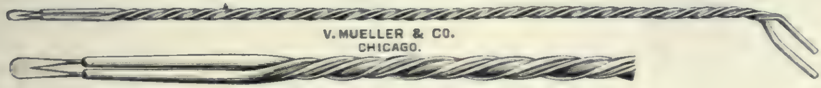

FIGURE 11.

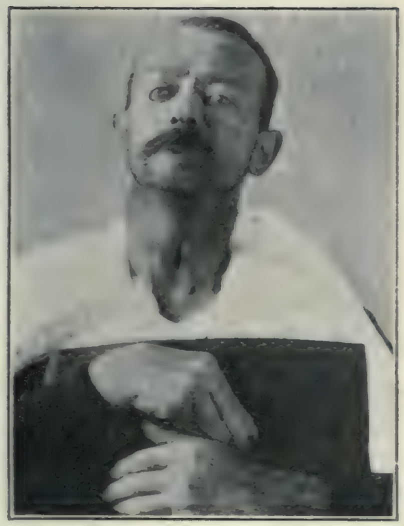

FIGURE 12.

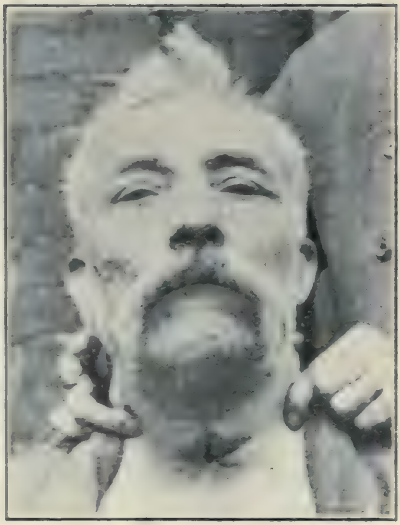

FIGURE 14.

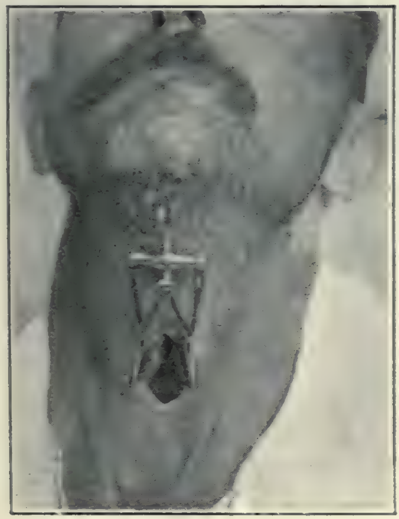

FIGURE 13.

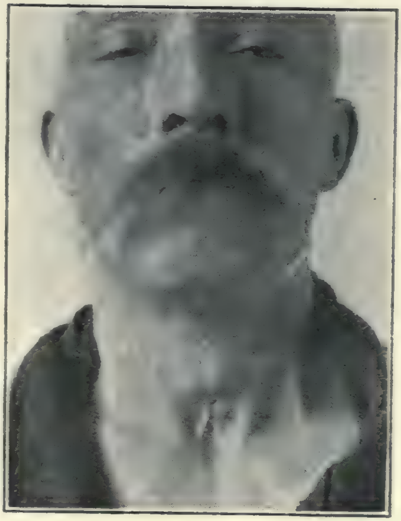

FIGURE 15

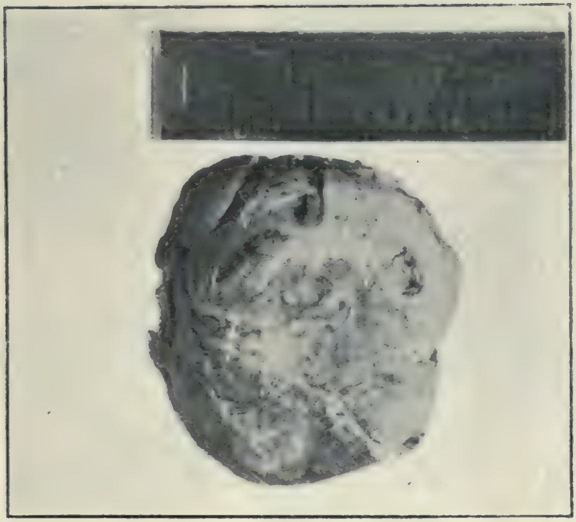

FIGURE 16.

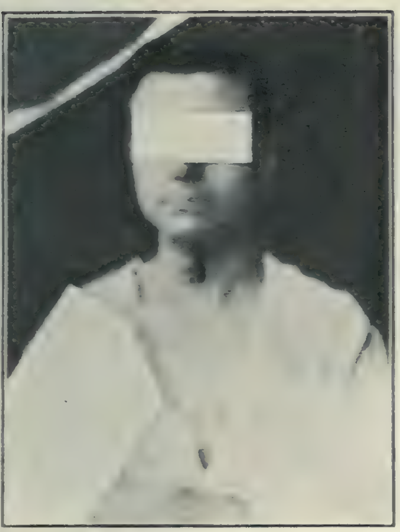

FIGURE 17.

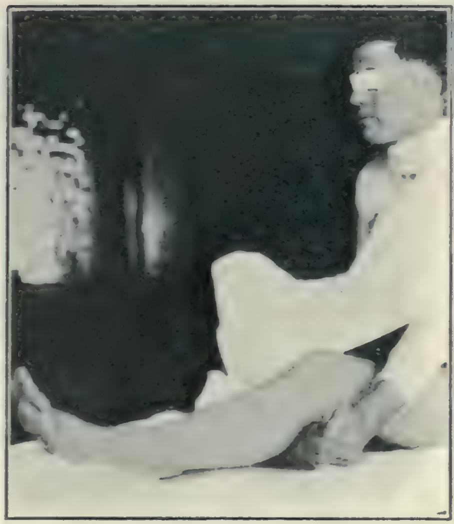

FIGURE 19.

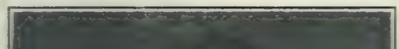

FIGURE 18.

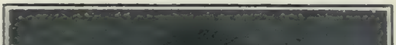

FIGURE 20.

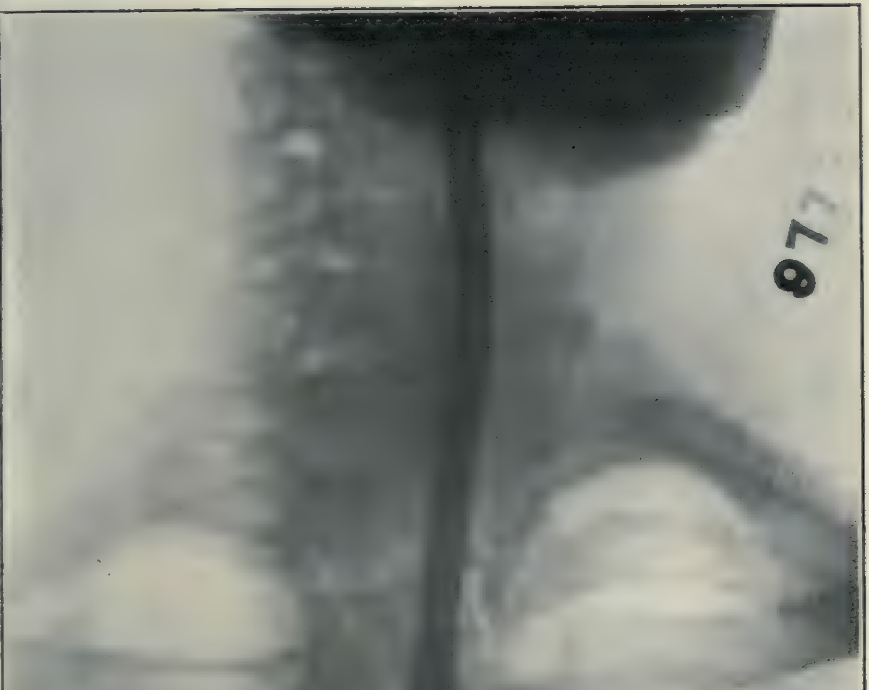

FIGURE 21.

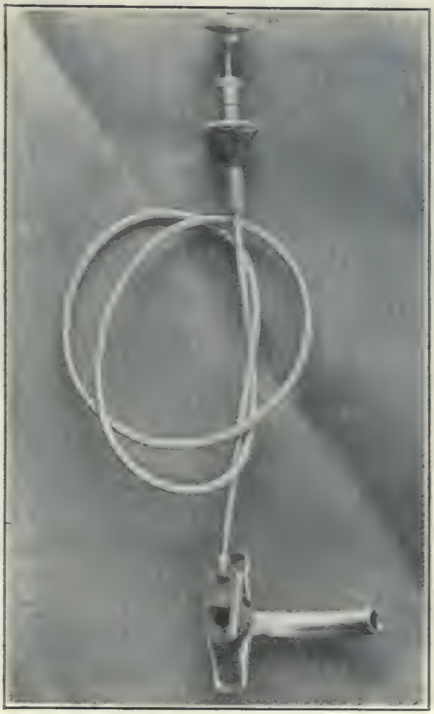

FIGURE 22.

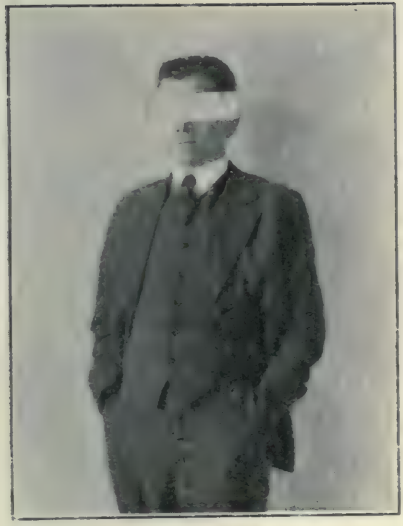

FIGURE 23.

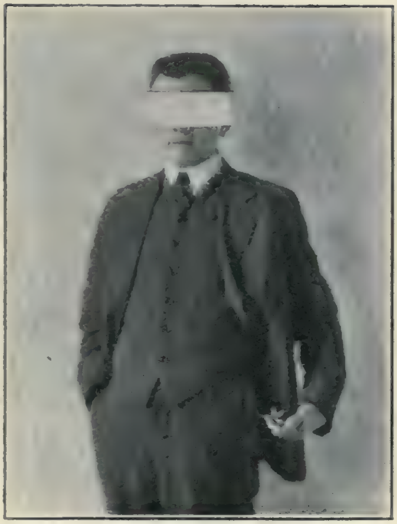

FIGURE 25.

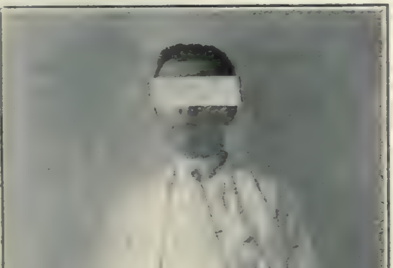

FIGURE 24.

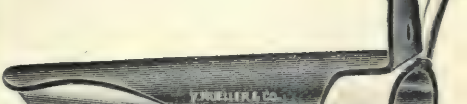

FIGURE 26.

XII.

THE GRAM STAIN IN MAKING A BACTERIOLOGIC DIAGNOSIS IN OTOLARYNGOLOGY.

By A. B. Bennett, M. D.,

Washington, D. C.

When vaccin therapy was first suggested in ear, nose and throat practice, I thought it an impractical idea unless associated with elaborate laboratory work, owing to the extensive bacterial flora found usually about the mouth and throat. Later on, Dr. E. R. Stitt, U. S. N., suggested that I try a routine Gram stain on the secretions of ear, nose and throat cases, and determine if it would not prove a valuable aid in making a bacteriologic diagnosis. I have followed the suggestion, and take pleasure in presenting a synopsis of this work on over one hundred and sixty cases, which includes a microscopic study of over three hundred Gram stained smears.

The Gram stain is especially adapted for a rapid determination of the bacterial contents of secretions from the ear, nose and throat. It takes but a few minutes to prepare the specimen for the microscope, and it differentiates to a great degree of accuracy the organisms found. Gram positive bacteria are those which retain the gentian violet after decolorizing and counterstaining, and Gram negative bacteria are those which do decolorize and take on counterstain.

The bacteria which are Gram positive are the staphylococcus aureus, albus, and pyogenes, streptococcus, M. tetragenus, pneumococcus, anthrax bacillus, tubercle bacillus, lepra bacillus, tetanus bacillus, diphtheria bacillus, diphtheroid bacillus, B. aerogenes capsulatus, odium albicans and mycelium of actinomyces, saccharomyces, Hoffman's bacillus, and B. xerosis.

The Gram positive bacteria which may be considered important from an ear, nose and throat standpoint are the staphylococcus, streptococcus, M. tetragenus, pneumococcus, tubercle bacillus, and diphtheria and diphtheroid bacillus. Each of these can be recognized from its characteristic morphology. The microscope cannot differentiate between the staphylococcus aureus and albus, but such differentiation is not important. The recognition of the tubercle bacillus would have to be confirmed by the acid fast test, and the B. diphtheria cannot be distinguished from the B. diphtheroid, as both have a changing morphology from dotted to oblong segments, and, according to some eminent bacteriologists, even the guinea pig test is not absolute as a point of differentiation.

The Gram negative bacteria are the meningococcus, M. catarrhalis, M. melitensis, B. typhosus, B. coli communis, B. dysenteriæ (Shiga), spirillum choleræ asiaticæ, B. pyocyaneus, B. mallei, B. pneumoniæ, Friedlander group, B. proteus, B. influenzæ, B. of bubonic plague, B. of chancroid, B. of Koch-Weeks, and the gonococcus.

The Gram negative bacteria which may be considered as important from an ear, nose and throat standpoint are the M. catarrhalis, B. pyocyaneus, the Friedlander group and B. of influenza. Here again recognition becomes fairly easy, as the only Gram negative coccus is the M. catarrhalis. The B. pyocyaneus, while capable of many forms, is usually seen as a rod shaped organism very similar to the typhoid or colon bacillus, which distinguishes it under the microscope from the Friedlander group, which show a distinct capsule. No bacteriologist will admit the absolute diagnosis of an organism until it has been cultured and tested.

In the early part of this work I had all the secretions cultured, also occasionally throughout the year, and the smear diagnosis and the culture diagnosis have agreed with unfailing constancy. However, it often happened that in a mixed infection the culture would show a different predominating organism than the smear, due to the fact that the predominating organism in the discharge was a poor grower on artificial media and was outgrown by a less numerous organism which happened to be a good grower on artificial media. In many laboratories a smear is considered an essential control of a culture in order to make sure that the predominating organism in the pus has not been outgrown by an unimportant organism which is in the pus or has been accidentally introduced. Another important condition shown by the stained smear is phagocytosis, which is an excellent factor in determining the active infectious organism when confronted with a mixed infection, and it also indicates the resistance of the patient against the organism.

I have grouped this series of about one hundred and sixty cases by diseases, and will briefly summarize the findings under each head.

DISEASES OF THE NOSE AND THROAT.

There were seventy-six cases of acute catarrhal sinuitis, one or more cavities involved, and of different degrees of intensity, but all showing on irrigation of the nose the characteristic catarrhal sinus secretion. The smears were all made from these secretions, taken directly from the nose with no possibility of mouth contamination.

In twenty cases the first smear showed no organism, but five on subsequent examinations showed the presence of bacteria, and nine remained free from microscopic evidence of infection, indicating that probably such a condition as a purely noninfectious acute catarrhal sinuitis does occur. In six there were no subsequent examinations made. These cases made very prompt recoveries under simple local treatment.

In forty-two cases (fifty-seven and one-half per cent of the total number) there was a single or pure infection—twenty-two with the pneumococcus, fourteen with the staphylococcus, and six with the M. catarrhalis.

In fourteen cases there was a mixed infection—five had the pneumococcus predominating and phagocytized, two had the staphylococcus, one had the Friedlander group, and one the streptococcus predominating and phagocytized.

Six cases showed the bacteria in about equal numbers—the diphtheria bacillus and the staphylococcus in one, the pneumocuccus and the staphylococcus in four, the M. catarrhalis and staphylococcus in one.

In one case the first smear showed a pure staphylococcus, and one month later the secretions showed only the pneumococcus and the M. tetragenus.

Many of these cases were clinically la grippe or epidemic influenza; four cases due to a pure pneumococcus infection were from one house epidemic; four cases due to a pure staphylococcus infection came from two house epidemics, two cases coming from each house. The main points in this series is that the great majority of the cases of acute catarrhal sinuitis are infectious, that over fifty-five per cent were due to a single or pure infection, and that the pneumococcus and the staphylococcus were the most frequent causes.

There were ten cases of chronic catarrhal sinuitis involving one or more cavities, having existed for long periods of time and exhibiting on nasal irrigation the characteristic catarrhal thick yellow or yellowish green heavy secretions. The smears and cultures were made directly from these secretions, without permitting any mouth contamination.

In two cases there was a pure staphylococcus infection, and one case of mixed infection, where the staphylococcus predominated and was phagocytized, although the pneumococus, M. catarrhalis, Friedlander group, B. pyocyaneus and the M. tetragenus were also present. Another case had the same multiple infection without any one organism predominating.

In two cases the B. pyocyaneus predominated, with the pneumococcus and M. catarrhalis present.

Two cases had only the pneumococcus and M. catarrhalis, and another only the pneumococcus and Friedlander present. The last of these cases I will report in greater detail, as follows:

A young man, with a history of chronic catarrhal sinuitis of three years' duration, accompanied by hoarseness and severe cough and expectoration, occasional night sweats and no loss of weight. There was a tumor on the left thyroid cartilage, obscuring the left vocal cord. The first smear from the sinus secretions had the pneumococcus predominating and phagocytized with the M. catarrhalis; streptococcus and staphylococcus also present. The sputum smear proved a pure pneumococcus infection markedly phagocytized. I gave him two injections of pneumovaccin, which was followed by a decided improvement and a practical disappearance of sinus symptoms. I also made a smear from a swab applied directly to the laryngeal tumor, and it contained only pneumococci, and no tubercle bacilli. I gave him deep inhalations of medicated oil, which lessened the cough and expectoration, with no change in the tumor. I again examined the sputum and found abundant tubercle bacilli with only a few pneumococci. I continued the oil inhalations for several weeks, the patient improved generally—gained weight, and the cough and the expectorations practically ceased, the last sputum examinations showing no tubercle bacilli and but a few pneumococci. The laryngeal conditions remained the same; the patient then moved to another part of the country. In this case the active pneumococcus infection obscured the tubercle infection, which was revealed on relief of the acute inflammation. In these cases of chronic catarrhal sinuitis a multiple infection seems the rule, and the staphylococcus, and the B. pyocyaneus are the most frequent offenders.

There was only one case of acute suppurative sinuitis, and that occurred in the right maxillary sinus. Pulsating pus was flowing into the middle meatus, and other cardinal symptoms were present. The smears and culture showed a pure staphylococci infection. I gave five injections of an autogenous vaccin along with local treatment, and obtained a quick recovery.

There were nine cases of chronic suppurative sinuitis with free pus flowing from the infected cavities. Two were pure infection, one being pneumococcic and the other B. pyocyaneus. Two were of dental origin—in one of these the smear exhibited a pure staphylococcus infection, and the other a mixed infection of M. catarrhalis, B. pyocyaneus, B. diphtheriæ, pneumococcus, staphylococcus, and streptococcus. In one case there was also caries of the ethmoid bone, and the smear showed a mixed infection, the streptococcus predominating and phagocytized with the M. catarrhalis, Friedlander group, pneumococcus and the fusiform bacillus also present. Another case contained staphylococci, streptococci, pneumococci, and B. pyocyaneus, and another case contained pneumococci, staphylococci, streptococci, M. catarrhalis and the B. influenzæ. In the last of these cases the first smear revealed a pure streptococci infection with marked phagocytosis. Later there was, in addition to the streptococci, a few staphylococci; four months later the infection appeared principally staphylococcic with a few streptococci, and a few weeks later the streptococci and staphylococci were in equal amount and there appeared a few B. pyocyaneus, the culture showing staphylococci and streptococci, each forty per cent, and the bacillus pyocyaneus, twenty per cent.

There were sixteen cases of chronic nasopharyngitis. The smear from one contained only an epithelial débris with no organisms. The others were all mixed infections, with pneumococcus predominating in nine, and the bacillus pyocyaneus being very numerous in nine. The other organisms found were the staphylococci, streptococci, Friedlander group, M. catarrhalis, diphtheroid bacillus and M. tetragenus. One of the features of the smears was the presence of large epithelial cells. Some of these were specimens which were claimed to come from other sources, but the picture was typical of a nasopharyngitis. In five cases I used mixed vaccin as indicated by the microscope, and in four there was decided benefit.

There was one case of acute nasopharyngitis. The smear revealed a pure staphylococci infection, and a culture contained a pure growth of the staphylococcus albus. Later the inflammation extended to the sinuses, and the smears from the sinus secretions contained staphylococci and pneumococci.

There were five cases of atrophic rhinitis. In three the smear contained a mixed infection of diphtheroid bacillus, staphylococci, M. catarrhalis, M. tetragenus, pneumococci and B. pyocyaneus. In the fourth case the smear contained only the diphtheroid bacillus and B. pyocyaneus. In two cases I used an autogenous vaccine of all the organisms found in the discharge. One case received thirteen injections, with slight improvement. The other case I have given over thirty-one injections, the dose increasing to 2,000 million; the local improvement was marked and the improvement in general health was quite remarkable.

A smear from an ulcer of the nasal septum contained principally a fibrous exudate, with only occasionally a single pneumococcus and a staphylococcus—really no infection. This ulcer proved to be due to syphilis that had been contracted thirty years before.

One patient complained of a nocturnal accumulation of pus in his throat and mouth. His nose, throat and larynx were normal. A specimen of pus brought by the patient corresponded exactly with the pus expressed from suppurating gums about two molar teeth, and contained pneumococci, M. catarrhalis, bacillus pyocyaneus, Friedlander group, leptothrix and spirocheta.

In one case of acute tonsillitis, where a thin membrane had extended over the posterior pillar, I peeled off a strip of the membrane and the smear contained pneumococci predominating with streptococci, staphylococci, M. tetragenus, M. catarrhalis, and a long search revealed no diphtheria bacilli.

In examining sputa from the larynx, trachea and bronchi, I always washed the secretions in sterile water, to remove the saliva and its possible bacteria before smearing it on the slide. There were two cases of acute laryngotracheitis. In one there were no organisms, and in the other there was a mixed infection, the staphylococci, pneumococci, diphtheria bacillus, M. tetragenus, M. catarrhalis, Friedlander group, and B. pyocyaneus being present. In this case I gave a mixed vaccin with good result.

One case of chronic tracheitis contained in the smear the pneumococcus predominating and phagocytized with the M. tetragenus, M. catarrhalis, staphylococci and B. pyocyaneus also present. I gave the patient four injections of pneumococcus vaccin with a good result.

One case of chronic laryngitis due to habitual clearing of the throat was interesting in that I examined the sputum many times and always found no organisms present.

In four cases of chronic tracheobronchitis the smears all indicated mixed infections of pneumococci, staphylococci, streptococci, M. tetragenus, Friedlander group, B. pyocyaneus, and M. catarrhalis. I gave a mixed vaccin to two of these cases, and though they had been of long standing there was a prompt response and recovery.

There were two cases of laryngeal carcinoma, in each of which the smear contained all the organisms found about the mouth and throat, with numerous pus cells but no phagocytosis.

I have examined the pus from a great number of cases of chronic follicular tonsillitis, and the microscopic picture was invariably the same, showing a mass of different organisms, among them being the pneumococcus, staphylococcus, streptococcus, M. tetragenus, M. catarrhalis, diphtheroid bacillus, B. pyocyaneus, Friedlander group, leptothrix, spirocheta, and fusiform bacillus.

DISEASES OF THE EAR.

In cases with aural discharge the microscopic examination is much more satisfactory than with discharge from the nose and throat, for the reason that contamination is practically impossible. Of course, if the smear is made from pus lying in the auditory canal, there is a possibility of other organisms being present from the skin about the canal. But if the canal is first wiped dry, the ear inflated, and the pus which flows from the middle ear through the perforation is caught up on a sterile cotton swab and a smear made, there is certainly very little chance of other than the organism really in the discharge being present.

There were five cases of acute suppurative otitis media. In two the infection was a pure staphylococci infection, one was a pneumococcus, and one was a pure infection of the diphtheria bacillus; and the last case was very acute, having violent pain, rupture of the membrana tympani, and an outflow of sero-sanguineous fluid, a smear from which failed to reveal any organisms—this case ceased to discharge on the second day. The first two cases also made rapid recoveries under simple local treatment.

There were six cases of acute suppurative otitis media with mastoiditis. Three cases showed a pure pneumococcus infection, two made complete recoveries under the use of pneumovaccin and local treatment, and one was operated upon. Another case was of a few weeks' duration, the external plate of the mastoid had ruptured, and there was fluctuating pus under the integument. The smear contained a tremendous number of streptococci and fusiform bacilli. I did a mastoid operation immediately, and in two weeks made a smear from the wound, and there were no organisms present. In the fourth case the conditions came on as a complication during an attack of measles. The smear showed a pure streptococcic infection, with a high degree of phagocytosis. The patient was desperately ill. I used streptococcus vaccin with decided benefit, followed by recovery. There was an apparent closure of the perforation and hearing power for the watch of 16/30. The perforation evidently opened, for one year later the child's mother dropped into this ear some "sterile" boric acid solution, followed by an acute suppuration, the smear indicating a pure diphtheroid infection. There was a quick recovery under simple local treatment. Four months after this attack the patient was bathing in a swimming pool and there was again an acute suppuration from that ear, the smear showing a pure staphylococcic infection. Simple local treatment again produced a quick recovery. In another case the M. catarrhalis was the infectious agent, and the case went to operation. It is interesting to note that in the ten cases of acute suppurative otitis media where an infection was found, it was always a pure infection, and that in one case with three different attacks, it was a pure infection of a different organism each time, and not the breaking out of the first infection, as one might readily suppose.

There were sixteen cases of chronic suppurative otitis media. The suppurations had been of six months' to thirty years' duration. In two cases there was a pure diphtheroid infection. In three cases there was a pure staphylococcus infection, in one of which there was a marked phagocytosis; in another there was also a Gram positive fungus present; and in the third there was a prompt cessation of discharge after using vaccin; later the discharge reappeared and was different in character than before, the smears and culture showing a pure infection with the B. pyocyaneus. In four cases the B. pyocyaneus was the active cause. One of these cases was of thirty years' duration; both the smears and culture showed a pure infection, and I gave eleven injections of autogenous vaccin, followed by complete recovery. Another case, also of thirty years' duration, contained on the smear a few diphtheroid bacilli and staphylococci, with the B. pyocyaneus predominating—the culture was pure B. pyocyaneus. I administered eleven doses of an autogenous vaccin, and the ear has been absolutely dry for eighteen months. The third and fourth cases merely showed the B. pyocyaneus as predominating, with a few diphtheroid bacilli present. In two cases the M. catarrhalis was the only organism found in the pus taken from the middle ear; however, in one of these the first smear of pus taken from the canal showed a few diphtheroid bacilli with the M. catarrhalis. Both of these cases made early recoveries under local treatment. There were three cases of mixed infection, two with the B. pyocyaneus, diphtheroid bacilli, staphylococci, and the other with B. pyocyaneus, diphtheroid bacilli, M. catarrhalis, and M. tetragenus.

If I may take the time, I would like to report a case in detail:

W. D. F., male, white, married, aged thirty-one years. March 29, 1912. Diagnosis: Chronic suppurative otitis media, right; chronic suppurative otitis media, residual, left. Duration: Intermittently for twenty-nine years. Membrana tympani, right, absent; membrana tympani, left, scarred and adhered to promontory. Smears and culture from right ear indicated a pure B. diphtheriæ infection. Administered seven increasing doses of autogenous vaccin. The discharge ceased shortly after beginning treatment, but I continued the vaccin, hoping to establish an immunity from a recurrence.

October 18th, six months later, the patient reported, with right ear still dry, but having a discharge from the left ear. A smear revealed pure bacillus diphtheriæ infection. I used all manner of antiseptics in the ear, including solutions of the silver salts and of iodin, until November 14th, during which time frequent smears showed a steadily increasing number of diphtheria bacilli—the last smear showing a solid mass of the organisms. I then blew into the ear some acetanilid and boric acid powder, which immediately stopped all discharge.

On December 19th, one month later, the right ear recommenced to discharge, accompanied by pain and throbbing; the discharge contained bacillus diphtheriæ and staphylococci in equal number, and both phagocytized. The left ear had also started slightly, and a smear from that side contained a few staphylococci.

On January 4th he reported, with right ear discharging, and a smear contained a large amount of B. diphtheriæ. He also was suffering with an attack of acute sinuitis, and a smear from the sinus secretions contained B. diphtheriæ and staphylococci. Five days later he was practically well and the ears were dry. I then examined the pus from his tonsil crypts, and found the leptothrix, B. pyocyaneus, M. catarrhalis, staphylococci, streptococci, pneumococci and a few B. diphtheriæ.

On January 22nd he reported, with a severe cold and the right ear discharging freely. The smears contained a few staphylococci and B. diphtheriæ, both phagocytized. I gave him three doses of a stock staphylococci vaccin, and on February 3rd the right ear was dry, but the left ear was moist, a smear showing a pure staphylococcus infection. He then passed from observation.

In this case there was no immunity produced by the diphtheria vaccin to a reinfection by the B. diphtheriæ, which was to be expected. The best fluid antiseptics were of no avail, while powders blown into the ear proved of great value; and lastly, the three doses of staphylococcus vaccin had not made much impression, as the left ear was being infected with the staphylococcus at the last observation.

There were five cases of chronic suppurative otitis media with mastoiditis. The suppurations had existed for a long period of time, and there was occasional pain and tenderness over the mastoid process. One case was in a negro baby, a neglected case, and the smear indicated a streptococcic infection, with the staphylococcus, M. catarrhalis and the fusiform bacillus also present. The second case, of twenty-six years' duration, was a pure infection of the diphtheroid bacillus, and was quickly cured by simple local measures. The third case was of twenty years' duration, and the smear contained apparently a pure culture of B. pyocyaneus. The culture contained two organisms, one being an immobile pyocyaneus bacillus which agglutinated in the presence of the patient's blood. An autogenous vaccin was made and four doses given, with complete recovery. In the fourth case the suppurations had existed since an attack of typhoid fever in Porto Rico in 1898. The smear and culture contained about ten different organisms, one of which was culturally the B. typhoid. It was proven to be the typhoid bacilli, and it agglutinated in the presence of the patient's blood. I gave him typhoid vaccin, and there was a decided improvement. This case was an aural typhoid carrier. He passed from observation before I could make any further tests. The fifth case was of seven years' duration, the smears showed great numbers of staphylococci phagocytized and a few diphtheroids. The sixth case I will report in brief detail:

October 18, 1912. A. S. B., male, white, single, aged fifteen years. Chronic suppurative otitis media of ten years' duration. Mastoid process tender and often painful. Membrana tympani absent, and pus flowing from posterior superior quadrant of middle ear. First smear and culture contain a pure culture of B. pyocyaneus. Administered three doses of stock pyocyaneus vaccin, and second smear contained less B. pyocyaneus and a few M. catarrhalis and staphylococci. Gave four doses of stock pyocyaneus vaccin, and the third smear contained no B. pyocyaneus, and a few staphylococci phagocytized. On December 15th the fourth smear contained a few staphylococci and no other organisms. On December 31st the fifth smear contained the B. pyocyaneus and staphylococci. Administered two large doses of pyocyaneus vaccin, and on January 14th the sixth smear contained a marked increase in number of B. pyocyaneus, and culturally there were the B. pyocyaneus and staphylococci. Administered six doses of autogenous vaccin, and on February 14th the seventh smear contained a large number of the B. pyocyaneus and staphylococci. I gave him six more doses of autogenous vaccin, and on March 27th the discharge was much less, and the eighth smear showed no B. pyocyaneus and only a few staphylococci. I continued giving the autogenous vaccin until twelve more doses were given, and the ninth smear contained the B. pyocyaneus in great numbers and a few staphylococci. The discharge is much less than originally and the boy's general health is better, but in spite of eight doses of stock pyocyaneus vaccin and twenty-four doses of autogenous vaccin, of the B. pyocyaneus and staphylococcus mixed, the pus from the ear still contains B. pyocyaneus and staphylococci.

There was one case of mastoiditis unaccompanied by any middle ear symptoms at the time of my first examination. Four weeks before I saw her there had been an acute middle ear suppuration which entirely recovered, and the membrana tympani and hearing were normal. Ten days before I saw her a swelling appeared over the mastoid that had steadily increased, and at my first examination there was a large fluctuating mass over the mastoid process. The child's health was good, appetite normal, and there had been no fever for the past week. I performed a simple mastoid operation. A smear from the pus that flowed from the first incision contained a very few staphylococci, all phagocytized, and no other organism, which agreed perfectly with the clinical picture, that she had entirely overcome the infection and that the pus and débris of the infection were lying under the skin.

In these twenty-four cases of chronic suppurative otitis media the B. pyocyaneus was the chief offender, with the staphylococcus and B. diphtheroid running close seconds. Twelve were pure infections, and ten were mixed infections.

This report is made entirely from the record cards in my own practice, and it has taken some time to accumulate even this small amount of data. The lesson has been invaluable to me, and I am sincerely enthusiastic over this metrod of acquiring an additional fact in the diagnosis of my cases. If I may draw conclusions from the smears I have made and personally studied, and from the cultures that I have taken and which have been grown, plated and differentiated in the laboratories of the U. S. Naval Medical School and the National Vaccin and Antitoxin Institute, they are these:

1. The active organisms infecting the ear, nose and throat are the staphylococcus albus and aureus, streptococcus, M. catarrhalis, M. tetragenus, pneumococcus, B. pyocyaneus, B. influenzæ, Friedlander group, and the diphtheria and diphtheroid bacilli. Most of these can be recognized under the microscope by the Gram method of staining. The fusiform bacillus is readily recognized, also, and is rarely seen except when there is decay or caries present.

2. The spirocheta and leptothrix seem to be inhibited, or at least inactive, in these diseased conditions. When the spirocheta are associated with fusiform bacilli, with the absence of other organisms, the picture is of Vincent's angina.

3. The Gram stained smear is of great value when administering vaccins, because, in the majority of instances, there is either a pure infection readily recognized, or there is a predominance and phagocytosis of one organism to indicate the vaccin needed; also because the effect of vaccin on the bacterial contents of the discharge can be periodically observed.

4. The Gram stained smear is of great value throughout the course of any infection of the nose, throat and ear, to observe the lessening or increasing of the bacteria, and to detect a change of infection from one organism to another.

5. Lastly and most important is the conclusion that a study of the smear with a recognition of the type of infection gives a knowledge of the prognosis otherwise unattainable. For instance, an infection with the M. catarrhalis, even if it be chronic, is usually generally relieved by simple measures. An acute infection by the pneumococcus is easily relieved and responds well to vaccin therapy. Chronic infections due to the staphylococcus or B. pyocyaneus are less quickly relieved, often taking considerable time and patience. The staphylococci infections are materially affected by vaccin, while the results in using the pyocyaneus vaccin are less certain. The presence in the smear of pus of the fusiform bacillus usually indicates

a grave condition with decay or caries. As is well known, the fusiform bacillus can only be found by the smear, as it does not grow on artificial media. It is impossible to state in detail the various points in a smear that gives one a prognostic vision, but experience with the microscopic pictures, associated with the after-results of the disease, has, I am sure, placed me on the threshold of another knowledge about my cases. I can heartily recommend it as a routine procedure.

XIII.

AN IMPROVED METHOD FOR DRAINING THE TYMPANIC CAVITY IN PURULENT OTITIS MEDIA.*

By John Guttman, M. D.,

New York.

A great majority of diseases of the ear begin with an acute affection of the middle ear. Prompt and proper attention to this middle ear trouble will result in a cure in a very short time; but if this be neglected, it will lead to a functional disturbance, or may result in an intracranial complication, which may become dangerous to life. These facts make it imperative that we pay at least as much attention, if not more, to the acute affections of the middle ear as we do to rarer and less important diseases of the other parts of the ear.

While the etiology and pathology of the most of the middle ear affections, especially those of an inflammatory character, are fairly well known, the therapeutic measures applied in these affections are identical with those in vogue about fifty years ago. In acute catarrhal, or purulent inflammations of the middle ear, we perform a myringotomy with a lancet-shaped needle or with a small knife with a somewhat broader blade, usually without anesthesia, and only in exceptional cases with local or general anesthesia. We thrust the point of a lancet needle into the lower anterior or posterior quadrant, or into the most prominent or bulging part of the drum membrane. The slit in the drum membrane is only as broad as the thickness of the piercing needle. The length of the incision varies. It is through this opening that the viscid or creamy purulent secretion must pass. If the process is very acute, the force of the accumulated secretion widens somewhat the opening, and in the course of weeks, months or years the secretion is discharged through this opening. This surely is no ideal form of drain-

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***Read before the Section on Otology, New York Academy of Medicine, January 14, 1916.**

age. No surgeon would consider this form of evacuation of pus from a closed cavity satisfactory. The narrow slit in the drum membrane made by the very thin needle or knife will in some cases close in a few hours, either on account of agglutination of some blood, or because some viscid secretion will fill out the small opening of the drum membrane.

To improve the drainage, which is the main therapeutic measure in all acute or subacute cases of purulent or catarrhal inflammations of the middle ear, I devised this little instrument.

This is a hollow steel barrel eight centimeters long, whose upper part is surrounded by a somewhat thicker steel mantel, with long furrows for the purpose of having a stronger and rougher grip in turning the trephine. At the lowest part there is a very sharp circular knife two millimeters in diameter. It is made by Meyrowitz.

This trephine is placed in the lower posterior quadrant, irrespective of the bulging part of the drum membrane, as the drainage will always be better the lower the opening is in the drum membrane. If the drainage is good the bulging part of the drum membrane will disappear spontaneously in three or four days. With two or three turns of the trephine a hole is pierced through the drum membrane. If the trephine is very sharp, a piece of the drum membrane will be found in the hole of the trephine. Immediately after the opening of the drum membrane in cases of otitis media chronica, a pulsating viscid, colorless secretion will make its appearance through the wide opening, and the subjective relief of the patient becomes very marked. In many cases the patient will remark voluntarily: "Now my head feels much easier." The eustachian tube is then catheterized, in order to evacuate the pus from the tympanic cavity.

Up to the present time I employed trephining of the drum membrane in more than thirty cases, in most of them with excellent results. In the beginning, when my technic was not as good as it is at the present time, the results of the operation were not quite satisfactory. I will not trouble you with the details of my statistics.

In looking over the literature I found that Himley advocated the use of a trephine about one hundred years ago; not for the purpose of facilitating the drainage, but to improve hearing. As it failed in its purpose, and in some cases even caused death, probably because it was handled too roughly, the use of the trephine was given up, as that of a dangerous instrument. In the *Laryngoscope* of 1912, Robert Lewis advocates the use of a punch forceps to remove a portion of the drum membrane, in order to overcome the inadequacy of drainage which sometimes follows ordinary myringotomy in acute cases of otitis media. But inasmuch as he does not describe the punch forceps, nor the method of its use, whether he grasps the drum membrane at the edge of the perforation, or whether he gets a fold of the drum membrane in the grasp of the forceps, whether he uses anesthesia or not, the writer is unable to pass judgment upon this method of procedure.

Although it is not expected that trephining will in all cases replace the present form of myringotomy (by paracentesis) with a lancet-shaped knife, especially in cases where general anesthesia cannot be used, still in numerous well adapted cases of ear affections, especially in subacute or chronic purulent otitis media, where the drainage is insufficient, its superiority to the present form of paracenthesis of the drum membrane will be acknowledged.

XIV.

SARCOMA OF THE NOSE: REPORT OF A FATAL CASE WITH METASTASES IN THE CERVICAL GLANDS AND IN THE BRAIN.*

By J. Payson Clark, M. D.,

Boston.

The patient, Mrs. X., aged sixty-four years, was first seen by me on March 15, 1915, being referred by her physician, Dr. R. L. Toppan, of Newburyport. Three or four months ago she noticed a swelling about the size of a pea on the left side of her nose. It is now (March 15th) about the size of a small marble. It has not been painful at any time. About the middle of February she began to notice a little bleeding on blowing the nose. For the past week this symptom has been more marked and there has been a slight bloody discharge on her pillow in the morning. There is no specific history, and the patient has always enjoyed good health. Examination shows a small rounded tumor, over which the skin is freely movable, on the left side of the nose, just below the nasal bone. On the inside of the left nostril can be seen a dark, purplish tumor on the outer wall just above the vestibule and extending anteriorly to the cartilaginous septum, on which it encroaches slightly. The antra and frontal sinuses transilluminate equally and well. A small piece of the rather firm growth was removed for microscopic examination, the patient's consent to an operation having been first obtained, in case the histologic examination should show the growth to be malignant. The pathologist's report was as follows:

"March 20, 1915. The specimen consists of a small segment of tissue, arising from which is a round white mass measuring about three millimeters in diameter. Microscopically the tissue is composed almost entirely of irregular masses of epithelial cells, which are separated from one another by a connective tissue stroma. Along one border (surface) there is an inflammatory exudate. The cells in themselves, though entirely of the squamous type, vary in size and shape; but more particularly the nuclei in many cases contain mitotic figures. Opinion: The tumor is a squamous cell cancer.

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*Read at the Annual Meeting of the American Laryngological Association, May, 1916.

"(Signed) FRANCIS L. BURNETT, M. D."

March 24th. An operation was performed, under ether anesthesia, with the assistance of Dr. George O. Clark. A longitudinal incision about two and a half centimeters long was made through the skin of the nose in the median line. At the lower end the incision was continued to the left cheek in the depression above the ala. The skin flap was elevated and the growth was seen protruding between the lateral cartilage and the nasal bone. The tumor was then removed with a margin of healthy tissue. The nose was packed lightly with gauze and the skin sutured with horse hair. No external dressing. The wound healed uneventfully by first intention. There was some sinking of the left side of the nose, but the deformity was very slight, considering the amount of tissue which had been removed. There was a small perforation of the septum. Professor F. B. Mallory of the Harvard Medical School kindly examined the growth for me and made the following report:

"Microscopic examination of your tumor of the nose shows that it is rapidly growing and infiltrating, as was obvious in gross. The diagnosis is not so positive. There is no sign of differentiation on the part of the cells. It can be classified either as a carcinoma or possibly as a melanosarcoma, with little evidence of the formation of pigment. It would require the tissue from the primary operation to make a positive diagnosis. I only mention the possibility, because if it were sarcoma, metastases are much more likely to occur. Possibly Dr. Burnett has the sections which he made originally, and I am sure he would be perfectly willing for me to see them."

Dr. Mallory saw the sections from the first specimen later, but was, I believe, unable to make a more positive diagnosis.

May 22nd. No local recurrence. A gland, size of a bean, can be felt under the middle of the left side of the jaw. It is freely movable and not tender. On June 4th the gland had fully doubled in size, and it was decided to remove it. This was done, under ether anesthesia, on June 14. The pathologic report on the gland was as follows:

"June 25, 1915. The specimen consists of a moderately firm piece of tissue, measuring about 2 x 1.5 x 1 centimeters, and of a pinkish color. Microscopically it is composed almost entirely of spindle shaped fibrous cells, though there is an area of round cell at one border, and areolar tissue peripherally. Furthermore, while the spindle shaped cells do not vary a great deal in size or shape, their nuclei are often found in a state of mitosis. No epithelial cells whatever are apparent. Opinion: Although the tissue taken from the original tumor was determined as malignant, the metastasis gives a clearer conception of the cells; it is not a cancer, but a spindle (fibro) cell sarcoma. (Signed) Dr. F. L. Burnett."

Confirmed by Dr. Mallory.

No other glands could be felt at that time. The patient was next seen on July 7th. There was no local recurrence and no glands palpable. On July 30th there was no local recurrence, but a gland could be felt under the left sternomastoid muscle.

In view of the diagnosis of sarcoma, it was now determined to try the Coley treatment. This was carried out by or under the direction of Dr. Torr W. Harmer, and was continued for two months. The following account of the Coley treatment of this case and general comments are taken from a letter of Dr. Harmer's to me, dated March 2, 1916:

"When recurrence occurs in such cases and the toxins fail to influence this recurrence, further operation is usually unsuccessful. Regarding her (Mrs. X.'s) treatment under my care: I saw her August 10th, and gave her one-half minim of mixed toxins (Coley). Further treatments were as follows: August 12th, one minim; 16th, two minims; 18th, four minims; 20th, four minims. All injections were in the musculature of the upper back. All produced more or less local irritation; none caused marked reaction, although a few caused some elevation of temperature and chilliness. The treatments were then continued by Dr. Toppan of Newburyport, under my direction. He gave the injections every other day, then twice a week, reaching a maximum of fourteen minims. I then suggested that injections be given into the growth (meaning the gland under the left sternomastoid, J. P. C.), starting with a marked reduction in dose. The result with an injection of nine or ten minims was most distressing: marked fever, chill, and vomiting, with temporary increase in size of tumor. A second injection into the tumor resulted in an equally distressing experience, and the toxin treatment was discontinued."

Dr. Harmer has had a large experience in this treatment of malignant tumors, and I shall give, at the end of this paper, a reference to a paper of his on this subject.

I saw the patient again on October 16th. Her general health appeared excellent, but the gland under the sternomastoid muscle was apparently unchanged, and there was a small recurrence on the cartilaginous septum, just below the perforation. This was a disagreeable surprise to me. I had been afraid of a recurrence on the outer wall of the nose, where I had cut rather close to the growth near its lower border, but not at the septal border, where a wide margin of healthy tissue had been taken. This recurrence was removed, under local anesthesia, on October 20th.

The report on this growth was as follows: "The specimen consists of a small piece of mucous membrane bearing a nodule about three millimeters in diameter, some cartilage and a sliver of bone. In a section of the soft tissue the cells are of an irregularly shaped spindle variety, with leucocytes and round cells scattered among them; but there also are some epithelial cells forming glands in the tissue and a covering of the tumor. The spindle cells vary in size and shape; some contain large, others small, nuclei, and now and then they are in a state of mitosis. The epithelial cells, on the other hand, are quite regular in size and shape. Opinion: The tumor is evidently a recurrence, as it is a spindle cell sarcoma."

The patient was next seen on November 13th. As the gland under the sternomastoid was apparently unchanged, it was decided, after consultation with Dr. George O. Clark and Dr. Harmer, to do a complete glandular dissection of the left side of the neck. This operation was performed on November 18th by Dr. George O. Clark. The dissection was done en bloc, and every vestige of glandular tissue removed. It is interesting to note that many glands were found involved which could not be palpated before the operation. The patient went home at the end of a week. There was a slight infection at the lower angle of the wound, where the dressing did not fully protect it. With the exception of this small complication, the wound healed solidly by first intention. There was a moderate left facial paralysis and some hoarseness (left recurrent paralysis?). I did not have an opportunity to examine the larynx at this time.

The subsequent history of the case is taken from Dr. Toppan's letters. The first signs of mental disturbance appeared on the night of December 5th, with rambling, incoherent talk. The next morning she had a convulsive attack with clonic convulsions, general in distribution, accompanied by rolling up of eyeballs, loss of consciousness for fifteen to twenty minutes, followed by pallor, a weak, thready pulse, and no remembrance of the attack. Subsequently the pulse, temperature and respiration were practically normal. The urine was normal, and there was no bowel trouble. For two weeks, following this attack, she was very restless, constantly moving hands or feet when awake, talking incoherently and irrationally, and sleeping poorly. She would start a sentence, but was, apparently, unable to finish it (motor aphasia). Her mind, however, seemed capable of understanding what was said to her during this time. She would answer rationally "Yes" or "No," but seemed unable to form words. The blood pressure (systolic) rose gradually from 150 to 180, going down again to 130 three days before death. Dysphagia increased slowly until even liquids were swallowed with difficulty, but later this symptom was not so marked. She lost weight and color. Reflexes were all apparently normal at first, and pupils reacted equally. She was drowsy at times and would sleep for the better part of two or three days, then wake and be very restless and irritable. About January 1st she practically ceased speaking and was more and more drowsy. It now became very difficult for her to walk, and by January 15th she was practically bed-ridden and had incontinence, worse of the bladder than rectum. There now appeared an exaggerated right knee jerk, with slight ankle clonus and some spasticity of the right arm, with twitching of right hand and arm and foot and leg. The right pupil was at this time slightly larger than the left, though both reacted somewhat sluggishly to light and distance. She became very comatose and hard to rouse, but would occasionally recognize her family. On January 17th the pulse became variable, ranging from 80 to 120 on different days. On January 22nd she sat up in bed and appeared very bright, but could not utter an intelligent word. On February 1st the right knee jerk was absent, and there was beginning atrophy of the muscles of the right hand and arm, which she does not move, nor is there any twitching in it now. She ate practically nothing for the last ten days of life and died on February 4th; the immediate cause of death being, apparently, myocardial insufficiency.

In a letter dated February 6, 1916, Dr. Toppan says: "Am enclosing a rough sketch showing the general location of the masses taken from the brain of Mrs. X. Those shown at the base of the brain were directly under the pia mater, about equal in size, much firmer in consistency than the brain substance and easily shelled out from the brain with the finger. The mass shown in cross section in the left internal capsule was softer and looked almost necrotic in its center; it also was easily shelled out, was roughly rounded and about three-fourths of an inch in diameter. As a whole, the brain was rather 'wet.' So far as I could see there was nothing to indicate the course of metastasis. Vessels seemed normal everywhere." Dr. Toppan found no evidence of local recurrence. He sent the specimens to Dr. Burnett for examination, whose remarks and opinion on these growths follow:

"The specimen consists of three evenly rounded, soft masses in formalin; one measures about 2 x 2.5 x 2.5 centimeters, and was taken from the anterior portion of the temporal lobe on the left side; another from the internal capsule on the same side measures 2.5 x 2 x 2 centimeters, and the third from the cerebellum, 2 x 1.5 x 1 centimeters. In sections the tissue consists almost entirely of spindle shaped cells, in which a nucleus in a state of mitosis is occasionally apparent. Vessels are not especially numerous. Opinion: The tumors of the brain are sarcomata; and are evidently secondary growths from the tumor of the nose.

"(Signed) DR. FRANCIS L. BURNETT."

Opinions confirmed by Drs. Graham and Mallory.

The very unusual and interesting features of this case are the metastases of the cervical glands and in the brain. While sarcoma of the nose often recurs again and again locally, metastases seem to be very rare. In the records of the Massachusetts General Hospital I find twenty cases diagnosed sarcoma of the nose. Twelve of these cases were admitted to the wards, and eight were treated as out patients. In none of these is mention made of metastasis. Since 1896 fourteen cases of sarcoma of the nose have been reported in the Transactions of the American Laryngological Association, in none of which metastasis occurred. On looking over the literature I have found but eight of nasal or nasopharyngeal sarcoma in which metastases are said to have occurred.

In 1911, T. H. Farrell reported a case in which there was a small cervical gland, but this was not examined microscopically

Adair Dighton reports a case in a boy, eight years old, which he saw first in June, 1913, in which there was a hard polyp like mass protruding from the left nostril, slight exophthalmos of the left eye, and prominence of the left cheek. There were small tumors in the roof of the mouth at the junction of the anterior and middle third of the hard palate. The submaxillary, sublingual and deep cervical glands were enlarged and hard. The boy had had tonsils and adenoids removed two years before, and about one year later the cervical glands were found enlarged. The nasal condition was not discovered until one month before the patient was seen by Dr. Dighton. On removal, both the nasal tumor and the cervical glands were found to be round cell sarcoma. Dighton is of the opinion that the adenoids and tonsils, removed two years before, may have been sarcomatous, as the glands appeared a year after this operation and before the nasal growth gave evidence of its presence.

C. N. Slaney, in October, 1914, reported a case of multiple round cell sarcoma originating in the nares. The patient was a man, aged forty-three years, with a history of syphilis and gonorrhea. He had had nasal obstruction for three years or more. There was a large pedunculated mass filling the nasopharynx, but more on the right side. The tumor, very friable, was removed by snare under local anesthesia. A year later there were pains in both knees and shoulders, with edema of the feet, and both nostrils were obstructed. Two months later a small painless fluctuating tumor appeared in the frontal bone. Five months later the patient fell, owing to spontaneous fracture of the right femur and left tibia and fibula. Other tumors appeared under the scalp and in the fingers. Five months later the patient died from obstruction of the trachea by a tumor. At the autopsy numerous erosions of the skull were found where the fluctuating tumors had been. There is no mention of a Wassermann test or of any syphilitic treatment, in spite of the syphilitic history. The microscopic report stated that the appearance was characteristic of round cell sarcoma.

It is well known that it is often impossible to differentiate a gumma from a round cell sarcoma by the microscope. The history of the case suggests that the multiple tumors were gummata rather than sarcomata. At all events, the diagnosis of sarcoma in this case is open to grave question.

J. A. Watson, in a study of one hundred and fifty cases, published in 1904, found metastases mentioned three times. In two of these cases the cervical glands alone were involved. These two cases have not been identified, as Watson does not mention them specifically.

Warthin's case, reported in 1899, the third case referred to by Watson, was a man, thirty-nine years old. A year and a half before seen by Warthin the left nostril became partly occluded, and a year later he had polypi removed from the nose which apparently were not considered malignant. For a month he had noticed numbness of the left side of the face. Diplopia, left sided ptosis, and dilatation of the left pupil soon followed. On admission there was weakness, nervousness, insomnia, and dull pains in the forehead. Nostrils were entirely occluded. The mind was clear. No operation was attempted. The condition became worse, and the patient died in coma six weeks after first seen. At autopsy a soft growth (size of a cherry) was found on the inner surface of the dura mater, near the longitudinal sinus. The pineal body and the hypophysis were replaced by tumor growth. The left third nerve and left Gasserian ganglion were completely surrounded by new growth. The soft tumor mass filling the nose was directly continuous with tumor masses filling the frontal sinus and sphenomaxillary fossa. The heart muscle was infiltrated by nodular tumors, and there was a large free tumor in the right ventricle. There were scattered small nodules in the lungs. All the abdominal organs showed metastases. The cervical lymph glands contained large tumor masses. The primary growth was a round cell sarcoma and the metastases showed a variety of pictures according to the age of the growth and the tissues in which it was located. The primary growth was in the nasal fossa, without reasonable doubt, according to Warthin. "The richness of the growth in cellular elements, the scanty stroma, the numerous large thin-walled blood vessels, the smallness and shape of the cells, all contributed to the facility of the rapid dissemination of the tumor."

In 1914, A. Finzi gives an incomplete report entitled: "A case of tumor of the nasopharynx with metastases in the base of the brain and in the spinal cord." In this case, a man, aged twenty-two years, with severe headache and nerve symptoms, there was a malignant process starting in the nasopharynx. Besides the possible metastases in the brain and spinal cord, the cervical glands were enlarged. No operation was apparently done, and, consequently, there is no microscopic report. As the patient was still living at the time of the report, the diagnosis of metastasis in the brain and spinal cord was made solely from the symptoms. The tumor was assumed to be probably sarcoma.

Sendziac, in an exhaustive article, published in 1913, on malignant tumors of the nasal sinuses and nasopharynx, does not mention metastases.

Ferreri, in the same year, in a paper on the treatment and prognosis of malignant tumors of the nasal fossæ and nasopharynx, says nothing of metastasis.

In a paper on the Coley treatment of inoperable sarcoma, published in 1915, Dr. Torr Wagner Harmer gives the following report: "Case 25.—Multiple small round cell sarcomata. M. C., housewife, fifty-five years old. December 7, 1912. Six months ago began to have severe frontal headaches. Four months ago, small hard swelling on right side of nose near bridge, which had gradually and painlessly increased in size. Three months ago, mass discovered in front of right ear, which has gradually and painlessly grown. About the same time a mass appeared just above the right clavicle and another in right neck. Behind right sternomastoid muscle, several pea to bean-sized glands. In front and behind left sternomastoid muscle, several small glands, also palpable. . . . Excision of several glands for diagnosis. (Description of the toxin treatment is omitted. J. P. C.) Patient died with metastases in abdominal wall, chest and both breasts, in four months."

Omitting as doubtful or not proven, Farrell's, Dighton's, Slaney's, two of the cases mentioned by Watson, and Finzi's cases, there remain only two cases besides my own in which the fact of metastases have been established, and only one with metastases in the brain. In my case the lymphatic channels through which the cervical glands became involved are quite evident. In describing the lymphatics of the face, Poirier and Charpy say: "The third group of lymphatics of the nose run with the facial vessels and terminate in the submaxillary glands. This cutaneous network is continuous with the lymphatics of the vestibule, and through them with the lymphatics of the mucous membrane. Anterior trunks of lymphatics run . . . . between the different cartilages, or even in front of them. They thus arrive at the celluloadipose subcutaneous tissue, where they unite into two trunks . . . . which terminate in the submaxillary glands."

Sobotta says that the submaxillary nodes empty into the superficial and deep cervical nodes. The natural course of the lymphatic stream thus accounts for the involvement of all the cervical glands in the anterior triangle of the left side.

The channel by which metastases reached the brain is not so clear. Quain's Anatomy says: "The lymphatics (of the nose) are abundant and large. They form a close plexus in the mucous membrane . . . . and a more open plexus of valved vessels nearer the bone. These are in communication with the lymphatic spaces which enclose the branches of the olfactory nerve, and these spaces again communicate with the subdural and subarachnoid spaces of the cranium so that the lymphatics of the nasal mucous membrane can be injected from the cranial cavity."

In the same connection, Poirier and Charpy say: "In the olfactory region of the mucous membrane of the nasal fossæ there are present, by the side of the lymphatic network, properly socalled, formations which are . . . . comparable to them. These are the meningeal sheaths of the first pair of nerves. Now, when these sheaths are injected through the subarachnoid space, it sometimes happens that the lymphatics of the nasal mucous membrane are filled at the same time. Axel Key and Retzius, who first noticed this fact, have never seen direct communications between the periolfactory sheaths and the lymphatics of the mucous membrane. . . . . Further study of the lymphatics of this region is important."

The fact that there were metastases in the meninges and brain in my case would seem to show that there may be a direct communication between the lymphatics of the nose and the interior of the skull. It would seem, from the rich lymphatic system of the nose, that metastasis in cases of sarcoma would be the rule instead of apparently the rare exception. What may be the reasons for this fact I am at a loss to surmise, nor do there seem to be any facts in the cases so far reported which throw any light on this question.

I desire to take this opportunity of thanking Dr. G. O. Clark for his surgical aid in this case, Dr. Toppan for his valuable contributions to the history, Dr. Harmer for his notes on the Coley treatment, and Drs. Burnett and Mallory for their careful pathologic reports.

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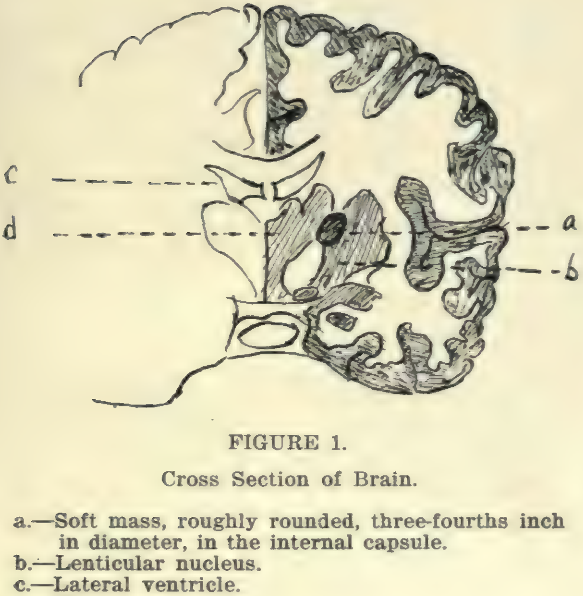

FIGURE 1.

Cross Section of Brain.

a.—Soft mass, roughly rounded, three-fourths inch in diameter, in the internal capsule.
b.—Lenticular nucleus.
c.—Lateral ventricle.
d.—Thalamus.

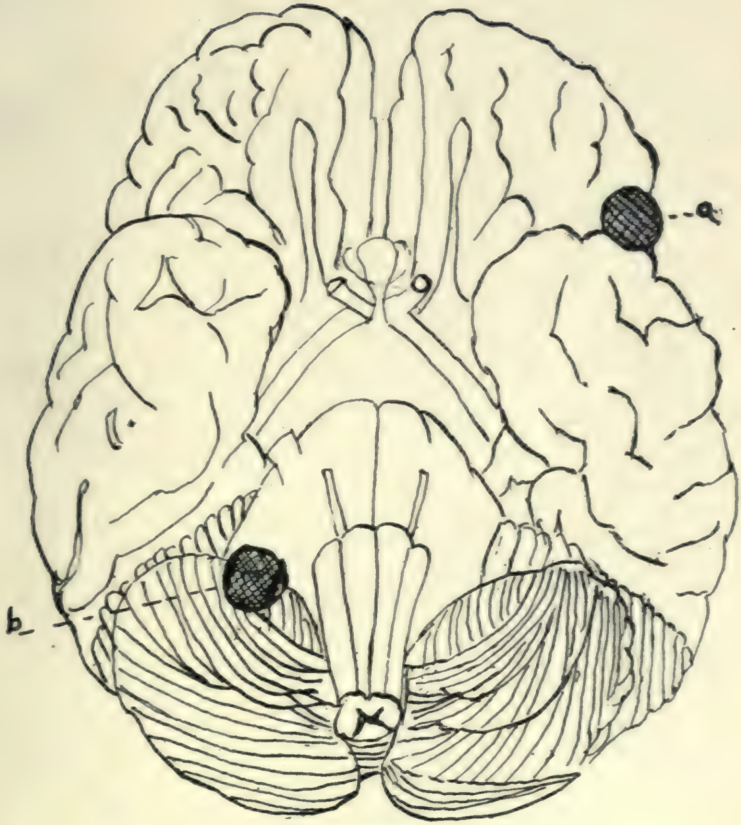

FIGURE 2.

a.—Round firm mass, one and one-half inches in diameter, at the anterior border of left temporal lobe at base of brain, just under the pia mater.

b.—Similar mass under the pia mater, just to right of olive, about where the vagus emerges.

XV.

DISSEMINATED MYELITIS AS COMPLICATION OF AN ACUTE MASTOID INFECTION.

By J. Ramsay Hunt, M. D.,

New York.

It is only too well known that cerebral complications occur very frequently after acute and chronic infections of the middle ear and mastoid region, in the form of inflammatory edema, meningitis, encephalitis and abscess. These are usually local manifestations, originating directly from the diseased focus and extending to adjacent areas of the brain substance and its coverings, through the blood and lymph channels, by direct contiguity from carious bone, or by traversing the sheaths of the facial and auditory nerves.

The central nervous system may also participate in a general pyemic process which sometimes occurs.

The form of infection to which I would call attention is one which, so far as could be determined by the clinical symptoms, was quite limited to the spinal cord—namely, an acute disseminated myelitis. This sequela of acute mastoiditis is of great rarity, if not unique in the literature of this subject, although it is well known that myelitis and encephalomyelitis

REPORT OF CASE.*

The patient is a woman, thirty-five years of age, a domestic servant by occupation. Her previous history is negative and without especial interest. She was in excellent health until April 1, 1915, when her right ear became painful following a "cold." The pain in the ear was very severe for two weeks, and was then followed by a purulent discharge from the canal. On the appearance of the discharge the pain diminished in severity. After the ear had discharged for about three weeks the flow suddenly ceased, the pains in the ear and mastoid returned and became increasingly severe, and she applied to the New York Eye and Ear Infirmary for treatment. She was admitted to the service of Dr. Gorham Bacon on May 11, 1915, a diagnosis of acute mastoiditis was made and operative treatment recommended.

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*Presented at the Clinical Conference of the New York Neurological Institute, November 9, 1915.

On May 13th Dr. T. L. Saunders performed a mastoid operation, which included exposure of the sinus and a considerable area of dura mater. Bacteriologic examination of purulent matter removed from the mastoid cells showed the presence of numerous colonies of streptococcus longus.*

On the fourth day following the operation she complained of indefinite pain in the back and lower extremities, followed by weakness and paresthesia of the trunk and legs. These symptoms were accompanied by fever and chilly sensations. Examination at this time showed some weakness and ataxia of the legs, and she was unable to stand or walk without assistance. Blood cultures made on May 18, 1915, were quite negative. The cellular constituents of the blood showed marked changes. Red cells, 2,200,000; white cells, 5,000; hemoglobin, 45 per cent.

Differential count as follows: Small mononuclears, 61 per cent; large mononuclears, 4 per cent; polynuclears, 34 per cent; eosinophiles, .5 per cent; mast cells, .5 per cent. The red corpuscles were irregular in size, with clear central areas. The urine was normal.

The pains in the lower extremities continued and soon extended to the neck and arms, especially on the right side. There was also a painful girdle sensation at the umbilical level, and a sense of constriction about the neck. Vesical symptoms and difficulty in defecation were also present. There was some headache low down in the occipital region, but no vertigo, delirium or other cerebral symptoms.

The mastoid wound healed without complication, but the spinal symptoms persisted and for a time increased in extent and severity.

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*Bacteriologic and other laboratory examinations were made under the direction of Dr. George S. Dixon.

Neurologic Examination.—On July 16, 1915, I first saw the patient, at the request of Dr. Bacon and Dr. Saunders. She was then almost paraplegic and unable to stand without assistance. There was marked static ataxia (Romberg symptom). She complained bitterly of pain in the neck and lumbar region, and there was a painful sense of constriction around the waist, with paresthesia of the hands and both lower limbs to the knees. The motor power of the arms was undisturbed, and there was no ataxia or tremor. The knee jerks and Achilles jerks were exaggerated, and there was a tendency to ankle and patellar clonus on both sides. The abdominal reflexes were elicitable, and the plantar reflexes were much diminished and of the flexor type.

There was marked disturbance of sensibility, both superficial and deep, of the lower extremities.

The pupils were equal and reacted to light and accommodation. Vision and the optic nerves were normal, and there was no nystagmus or paralysis of cranial nerves. Articulation was not affected.

At this time (June 16, 1915) a Wassermann test of the blood and spinal fluid was negative, and there was no increase of the globulin content, or of cellular elements. A blood count at the time showed some improvement over that taken on May 18th, but there was still severe anemia: Red cells, 2,160,000; white cells, 4,000; hemoglobin, 70 per cent.

Differential count: Small mononuclears, 53 per cent; large mononuclears, 10 per cent; polynuclears, 36 per cent. One nucleated red cell was found.

The diagnosis was made of a spinal cord affection, probably a myelitis of infectious or toxic origin. She remained in the infirmary until August, 1915, without material change in her condition, and was then transferred to the New York Neurological Institute, and was admitted to the Second Division (service of Dr. Frederick Peterson).

Her complaints on admission were as follows: Pain in neck and dorsal region, with broad girdle sensation about the chest and upper abdomen. Pain and painful paresthesia of the lower extremities and to a lesser degree of the arms, especially the right. Rather a constant area of pain in nape of neck extending to the right shoulder. Occasional incontinence of urine and also of feces after laxatives. Inability to stand or walk. There is no complaint of headache, vertigo, diplopia, or visual disturbance. Her mind is perfectly clear.

Examination.—August 8, 1915. The patient is bedridden and incontinent. No decubitus. Pupils are equal and react to light and accommodation. No nystagmus.

The cranial nerves, including the optic discs, are negative.

The spinal column shows no deformity, but there is some tenderness on pressure at the level of the fourth D. and eighth D. spines, and there is very slight stiffness of the neck. Movements of the arms are free, and there is no ataxia or tremor. The grip of the right hand is somewhat weakened, and the right arm is the seat of pain and paresthesia, but without gross disturbance of sensation. The tendon reflexes of the upper extremities are present and not exaggerated.

Both lower extremities are spastic and paretic, the right more than the left. There is well marked patellar clonus and ankle clonus on both sides. The plantar reflexes are of the extensor type (Babinski). The abdominal reflexes are absent.

The postural sense of the toes is disturbed on both sides, and the superficial sensibility, touch, pain and temperature, is diminished or lost in irregular areas below the umbilical line, above which there is a broad zone of hyperesthesia.

On August 8, 1915, the Wassermann tests of the blood and cerebrospinal fluid were again negative, and there was no increase of globulin or of cells. The urine was negative.

The blood on November 6, 1915, showed considerable improvement over previous examinations, viz.: 4,782,000 red cells, with 70 per cent of hemoglobin. While under observation in the Neurological Institute there had been no fever, and the examination of the internal organs, heart, lungs and abdomen, showed no abnormalities.

COMMENT.

The patient remained in the institute three months, during which time there was a gradual abatement of the subjective symptoms with some return of power in the lower extremities. When discharged, November 15, 1915, there was still spastic paraplegia with paresthesias and a girdle sensation. The spinal cord symptoms had, however, shown a distinct improvement, no new symptom had developed, and the tendency was to retrogression and not progression of the disease. There was, therefore, every reason to hope that the inflammatory process had been checked by the defensive forces of the body, and that eventually those neural structures which had suffered would gradually recover a considerable portion of their function. It is probable, however, that some weakness and spasticity will persist as a result of permanent injury to the pyramidal tracts.

As the spinal cord symptoms had followed so soon after the operative procedure, it was thought likely that the organisms (streptococci) or their toxins had entered some of the venous channels, thus reaching the general circulation. Just why the hematogenous infection should have become localized in the spinal cord is as much of a mystery in this case as in other of these obscure selective forms of inflammation. The possibility of a direct infection of the subdural space was also considered, with secondary invasion from the cerebrospinal fluid of the medulla spinalis. Such an invasion of the cerebrospinal fluid would be much more likely to cause meningitis, with the clinical and pathologic features of this disease, and while it is true that slight meningeal symptoms were noted, as spinal pain and some stiffness of the neck, these were not more acute than are commonly observed in myelitis, and the fluid obtained by lumbar puncture was free from any signs of inflammatory reaction.

The extreme degree of the infective and intoxication process is shown by the severe secondary anemia which was present. That this was merely a secondary anemia was evidenced by the marked improvement in the blood picture after the subsidence of the infection, and the funicular myelitis associated with pernicious and other grave anemias may, therefore, be excluded.

REMARKS.

This example of an acute disseminated myelitis is interesting and unusual because of the rare source of the infection—the middle ear and mastoid cells. The infectious myelitis, while not a common affection, is by no means infrequent, and may arise from a variety of causes. It is met with as an occasional complication after most of the acute infectious diseases of childhood, also affections like typhoid fever, osteomyelitis, erysipelas and during pregnancy and the puerperium. Not a few cases have been associated with infections by the gonococcus and prostatic abscess; and it has been observed after such mild affections as panaritium, tonsillitis and angina.[1] Of more than passing interest are those cases which have seemed to develop after inflammatory and suppurative processes in the abdominal cavity—viz., appendicitis, metritis, parametritis and septic peritonitis—as it has been shown by Orr and Rows[2] that infections may travel along the lymphatic channels of the nerve sheaths directly to the spinal cord. Oppenheim[3] and Flatau[4] have each observed myelitis as a complication of suppurative processes in the antrum of Highmore. Bacteriologic examination sometimes shows the presence of such organisms as streptococci, staphylococci and pneumococci, both in the cerebrospinal fluid and postmortem in the cord substance. Typhoid and tubercle bacilli have also been found, but as a rule bacteriologic studies are negative, and it is assumed that the organisms are either present in very small numbers or that they have been destroyed and removed by the leucocytes. It is more than probable that the toxins of such organisms play an important, if not the chief rôle in the production of these forms of myelitis.

In many of the cases the symptoms and the pathologic lesions are by no means limited to the spinal cord, and the brain stem, cerebellum and cerebrum are involved as well—the encephalomyelitis. A rare and very interesting type is the acute optic neuromyelitis, in which retrobulbar neuritis and optic neuritis with amblyopia and amaurosis are early manifestations.[5]

A few of the cases may terminate in suppurative meningitis and abscess formation, but, as a general rule, the infection takes the form of a nonsuppurative myelitis or encephalomyelitis, as is not rare when certain strains of pyogenic organisms reach the deeper structures in small numbers—e. g., infective arthritis.

The prognosis in these cases depends upon the virulence of the infection and the extent and location of the lesions produced. Many of the cases terminate fatally in the acute stage; in others, serious cord disturbances are produced and death results in the chronic period from bed sores, secondary bladder and renal involvement. A not inconsiderable number of cases recover completely or with slight motor disability.

An interesting group of cases is that in which periods of remission and intermission are observed, probably due to a recrudescence of bacterial activities. These constitute a borderline or transition type to the disseminated sclerosis.

In all those cases where it is possible to isolate an organism, treatment with autogenous vaccins should receive consideration.

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XVI.

BRONCHOSCOPIC AND ESOPHAGOSCOPIC POSTULATES.*

By Chevalier Jackson, M. D.,

Pittsburgh.

It has seemed to the writer that so many of the fundamentals of peroral endoscopy are, metaphorically speaking, in such a fluid state that the members of this society should formulate some sort of guidance for us all, to be modified to suit ourselves as applied to the particular case. The author is especially in doubt about the duration of bronchoscopy. Ingals, who is high authority, states that prolonged (over half an hour) bronchoscopies are dangerous. With this I heartily concur, but confess I, for one, do not know exactly where to draw the line. In the Pittsburgh clinic we are timing all our endoscopies, and hope eventually to accumulate valuable data. At present we try to work on the tentative basis of postulates three and four; but would be glad to have the views of others. Especially would we be glad if the safety limit of duration could be extended, because there are some difficult cases in which success seems so near that the utmost courage of one's convictions is required to desist and to postpone the bronchoscopy. Assuming that introduction of the bronchoscope does not require longer than one minute, all the time given is assumed to be devoted to bronchoscopic search and solution of mechanical problems of foreign body disimpaction and removal.

It is assumed that in recent, uncomplicated, untouched foreign body cases the mortality of bronchoscopy should not exceed two per cent. The simple passage of a bronchoscope in normal people as is said to be done in some clinics probably has no mortality.

These postulates are only tentative, to be discussed, rejected, accepted, excepted or modified, as experience may determine.

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*Read before the American Laryngological, Rhinological and Otological Society, May 4, 1916.

Though they are phrased dogmatically, the author makes no claim to finality, and therefore begs to call particular attention to his intention to offer these formulations simply as postulates for discussion, not as rules to follow, except in case of such of the postulates as may receive your sanction; and even then it must be remembered that all good rules have exceptions. I hope also to have you add any postulates to cover omissions that doubtless have occurred.

1. Anesthesia should be left to the choice of the operator in the particular case. Some operators will prefer general, others local anesthesia. Others none at all in children. Every laryngologist should do diagnostic direct laryngoscopy in children without anesthesia, general or local. Very few endoscopists use any anesthesia, general or local, for esophagoscopy in adults. For ballooning the esophagus, as used by Mosher, general anesthesia is needful. Ether is preferable to chloroform. Cocain and morphin are dangerous in very young children. A few endoscopists use novocain and alypin.

2. An aseptic technic should be carried out as thoroughly as if the abdomen were to be entered by external incision. While absolute sterilization of the field is impossible, cleansing of the teeth and rinsing of the mouth with dilute alcohol should be attended to whenever practicable. In any event, we never need to introduce any organisms other than those already harbored by the patient.

3. A bronchoscopy ordinarily should not be continued longer than twenty minutes in a child under five years of age; nor longer than thirty minutes in one under twelve years. Unless for urgent reasons, a bronchoscopy should not be repeated at shorter intervals than five days in children under five years of age. Thirty minutes should be the time limit of an esophagoscopy in children under twelve years. Both bronchoscopy and esophagoscopy may be done in the newborn, if necessary, but the duration should not exceed fifteen minutes, except in case of bronchoscopy for the maintenance of respiration.

4. Four or more very careful twenty-minute bronchoscopies, at intervals of a few days, in an infant, are practically harmless; while one of an hour's duration in an infant may be fatal, no matter how carefully done. This does not apply to adults, in whom there is no special danger attached to prolonged bronchoscopies.

5. There are no absolute contraindications to bronchoscopy or esophagoscopy in foreign body cases. Many conditions may render inadvisable endoscopy for disease, and in foreign body cases may justify postponement of the endoscopic procedure. Aneurism, if positively diagnosticated, is a contraindication to bronchoscopy or esophagoscopy for disease, but not for foreign body.

6. In very dyspneic cases, if there is any lack of confidence as to the promptness of a bronchoscopy, a tracheotomy is advisable. Then the bronchoscope should be inserted through the mouth, not through the tracheotomic wound. The peroral route gives more freedom of movement, greater ease of searching the anterior branch bronchi, and less danger of infection. It is the route whose every movement is familiar from practice. No one can tell, in looking or working through a bronchoscope, with any of the modern systems of illumination, whether the tube is a long or a short one. In a dyspneic foreign body case the foreign body may be in the larynx, whence it would be most easily removed perorally, anyway.

7. The primary requisite for safe bronchoscopy is gentleness.

8. A foreign body should not be pulled upon until the bronchoscopist has a clear idea of the mechanical problem, nor until he is certain that the foreign body is free to be moved without inflicting trauma. To pull upon a foreign body without first making sure that it is disengaged and free to be moved is to court both failure and disaster. The problem is not simply to remove the foreign body, but to remove it without serious danger to the patient.

9. It is dangerous to pull on a tack, pin, needle, nail or other sharp pointed object until the point is in the tube mouth. Even then it is unwise to pull strongly. A child is better off alive with a tack in its lung or esophagus than dead or dying with it out. In most cases of foreign body in the bronchi the patient will live many months if no trauma is caused by attempts at removal. Hence, hasty, desperate, ill studied, violent attempts at removal are unjustifiable.

10. Full-curved hooks are to be used in the bronchi with greatest caution, if used at all, lest they catch inextricably in branch bronchi.

11. Laceration or perforation of the thoracic esophagus is almost invariably fatal.

12. The watchword of the bronchoscopist should be, "If I can do no good, I will at least do no harm."

XVII.

CARTILAGE AND BONE IN THE TONSILS.

By W. E. Grove, M. D.,

Milwaukee.

Although our knowledge of the physiology of the tonsil is but fragmentary and incomplete, the pathology of this organ is very well known. We know that the tonsils are very often predisposed to infection, both acute and chronic, and comparatively seldom to tumor formation. Whether we shall class the formation of cartilage and bone in the tonsillar tissue as of neoplastic origin, or whether we shall regard such changes as due to the results of chronic infection, will depend largely upon our view as to the origin of such pathologic changes.

We know that the tonsil consists, in general, of collections of lymphoid tissue, arranged in follicles, grouped around crypts and separated from each other by trabeculæ of connective tissue derived from the fibrous capsule which surrounds it. This structure is covered on the exposed mouth side by a stratified pavement epithelium. These tissues, together with the blood and lymph vessels, form the histologic elements and tissues of which the tonsil is normally composed. When any other tissue, such as bone or cartilage, is found in the substance of the tonsil, it must be regarded as pathologic and must be explained as being either metaplastic or neoplastic in origin, or as being a misplaced embryologic rest.

As early as 1898 Walsham called attention to occurrence of cartilaginous and bony nodules in the tonsil. While making observations on the occurrence of the tubercle in the tonsil he came across two tonsils in which he found both bone and cartilage.

The first was from a man aged fifty years, dead of pulmonary tuberculosis. Both tonsils had undergone considerable atrophy. Scattered throughout the organ, at the base of the cypts, were numerous small masses of bone in the form of trabeculæ, rings or solid nodules. They did not encroach upon the adenoid tissue.

The second case was a man aged twenty years, dead of chronic pulmonary tuberculosis. In this case large masses of cartilage were scattered through the adenoid tissue. In some areas it had been converted into bone. The matrix was in some instances of hyalin, in others of fibrocartilage, variety. The nodules were surrounded by a dense fibrous tissue, forming a kind of perichondrium. Some of the nodules penetrated the adenoid tissue, in one instance almost reaching the epithelium.

These two cases were found in a consecutive examination of thirty-four postmortems.

Walsham assumes that these cartilaginous masses were embryonic rests from the fetal cartilage of the second branchial arch. Professor Kanthack, to whom he showed his specimens, dissented absolutely from this theory, and believed that the cartilage and bone formation were due to a metaplasia of fibrous tissue.

Wingrave, publishing in the same volume of the *Lancet*, first found such cartilaginous nodules in the microscopic examination of diseased tonsils, mostly suffering from a chronic hypertrophy, removed from patients under twenty years of age. This cartilage was both hyalin and white fibrous in type, imbedded in a dense connective tissue capsule, and never occurring in the lymph nodules or follicles. In one instance calcification was noted, but this he did not regard as truly osseous tissue.

In ten tonsils removed from cadavers, Töpfer found cartilage in all three times. He also found in a tonsil removed from a case of benign mycosis tonsillaris a number of areas of cartilage, always surrounded by connective tissue, and never in the midst of adenoid tissue. In one tonsil removed from an old man he found an area which he believed showed a direct gradual transition from the fibrillar connective tissue. The center of the cartilaginous area was hyalin. Around this was a transition zone, which he designates as "Netzknorpel," in which zone he believes the connective tissue was gradually being transformed into cartilage. He regards the hyalin cartilage as the finished product, and the fibrocartilage as a "vorstage."

Nösske found cartilage and bone in six cases; five times in both tonsils, and once in one tonsil only. The ages of these cases ranged from thirty-seven to seventy-six. These were found in various parts of the tonsil, but mostly in the deeper layers of the connective tissue septa. Numerous transitions from connective tissue to cartilage and bone could be made out. Nowhere did the cartilage or bone show any tendency to get out of the connective tissue. In all the cases atrophic and regressive changes were noted in the tonsils, mostly in the connective tissue septa.

Among others who have reported the finding of cartilage and bone in the tonsillar tissues are Schweitzer, E. Zuckerkandl, Imhofer, Munzer, and Reitman.

In order to determine whether we were dealing with an embryologic anlage of cartilage, Reitman examined in serial sections three human embryos of fifty-one, sixty and seventy-two millimeters in length. Furthermore, he examined serially fifty tonsils from thirty-seven individuals of all ages. In all, seventeen tonsils, or thirty-four per cent, gave a positive finding.

In none of the three embryos could he find anything definite, though in the embryo of sixty millimeters he found a piece of cartilage at the end of the styloid process, and in the position of the still undeveloped tonsil.

He found cartilage in the tonsils of four newborn infants, in one child of one-half year, in one of one and one-half years, and the other cases varied from six to thirty-three years. In most of these cases he had to deal with hyalin cartilage, less frequently with the embryologic type of cartilage, rich in cells. All of the cartilaginous foci were distinctly separate from any connection with the skeletal tissues.

My attention was first called to the subject by finding areas of cartilage in sections of the tonsils removed from a case of benign mycosis of the tonsils. The age of this patient was sixteen years. The tonsils were very small and atrophic, and the crypts were filled with the typical masses of mycelium of the fungus growth. The lymphoid tissue was greatly reduced in amount. The cartilaginous areas were found near the posterior capsule and in the connective tissue of the framework, having no connection with the lymphoid tissue. (Figure 1.) The cartilage was definitely hyalin in character at its center, and near its edges seemed gradually to pass over into the connective tissue of the tonsil by means of transitional fibrocartilage. (Figure 2.) No definite capsule could be made out, and at the edges of the cartilage, as in Figure 2, which is an enlargement of part of Figure 1, the direct transition from the connective tissue framework of the tonsil to the hyalin cartilage can be made out.

The second case, from my own practice, occurred about six months ago, in a young woman of twenty-three years, suffering from a chronic tonsillitis. The tonsils were slightly enlarged and the crypts were full of foul-smelling, cheesy plugs. The enucleation was very difficult, a snare wire being broken in removing the right tonsil. In passing the finger over the capsule of the tonsil the hard nodules could be plainly made out macroscopically. Sections of these tonsils show the numerous areas of cartilage. These are all of the hyalin variety. They are all situated in the connective tissue of the organ. They do not seem to have a very definite capsule, but one can make out the gradual transition into the fibrous connective tissue of the trabecular framework.

In running over the sections of the tonsils which we have removed at the Milwaukee County Hospital during the last two years, I found one other specimen. Undoubtedly, in working over this material we would have found many other instances had we made serial sections and examined every fifth or tenth section, as was done by Reitman.

CONCLUSIONS.

It would seem from these isolated cases, and from a study of the literature on the subject, that the presence of cartilage, and even bone, in the tonsillar tissue is not of uncommon occurrence. It would be found much more commonly and frequently were all tonsils removed, sectioned and systematically studied.

The cartilage found is true cartilage, and is of the hyalin and fibrocartilage varieties. It is uniformly found near the capsule or in the connective tissue framework of the organ. It never invades the lymphoid tissues. It has been found in all ages, but more usually in the tonsils of adults. It has most commonly been discovered in tonsils which were either undergoing regressive changes or which were the subject of chronic inflammatory changes; less frequently in the healthy normal tonsils of children.

Whether the bone tissue occasionally found in tonsils is true bone, or whether it is a calcification of preexisting cartilaginous tissue, is a question I cannot enter into with the material at my disposal. I am, however, inclined to think that true osseous tissue has never been found in the tonsil.

The theories offered to explain the presence of the cartilage are three: First, that it takes its origin from the fetal cartilage of the second branchial arch; second, that it is a remnant of misplaced embryologic rests; and third, that it is the result of a metaplasia of connective tissue occurring in tonsils which are the foci of chronic inflammatory processes, or which are in the process of atrophy.

If the cartilage occurring in these tonsils takes its origin from the fetal cartilage of the second branchial arch, or if it is a rest of embryologic cartilage, misplaced, we would naturally always expect to find only one variety of cartilage. As a matter of fact, we find two varieties of cartilage: fibrocartilage and the hyalin variety.

If the cartilage is a remnant or rest of misplaced embryologic cartilage, why should we uniformly find the foci only in certain regions of the tonsillar structure, and always bearing a definite relation to the connective tissue framework, as Töpfer points out?

Moreover, if the theory of misplaced embryonal cartilage be true, then we must expect to find the cartilaginous areas as frequently in the tonsils of children as in those of adults. This is not the case, for Nösske was never able to find these changes in children, and Reitman, although he did find the changes in children, was not able to find them as frequently in infants and embryos as in older children and adults.

It seems much more logical to me to explain these changes as a metaplasia or a metamorphosis of connective tissue, due to inflammatory, hypertrophic or atrophic regressive changes in the tonsil. The reasons for this are:

First, that the cartilage always occurs in definite relation to the connective tissue. This was true in my cases and in all the reports in the literature.

Second, these areas of cartilage never exhibit any true perichondrium about them, and can usually be shown to merge by gradual transition into the connective tissue surrounding them. This can be made out in my first case in Figure 2, which is an enlargement of a portion of Figure 1, where the hyalin cartilage first passes over gradually by means of fibrocartilage, and then a dense connective tissue rich in cells, into the normal connective tissue of the tonsil. In fact, we must consider the hyalin cartilage as the finished product in these cases, and the fibrocartilage as the "vor-stage" in its formation.

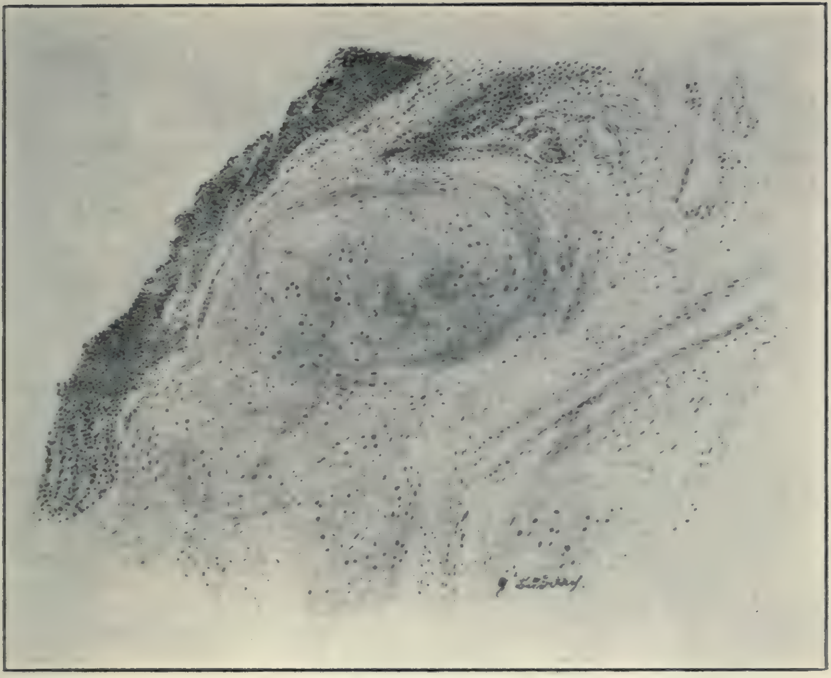

FIGURE 1.

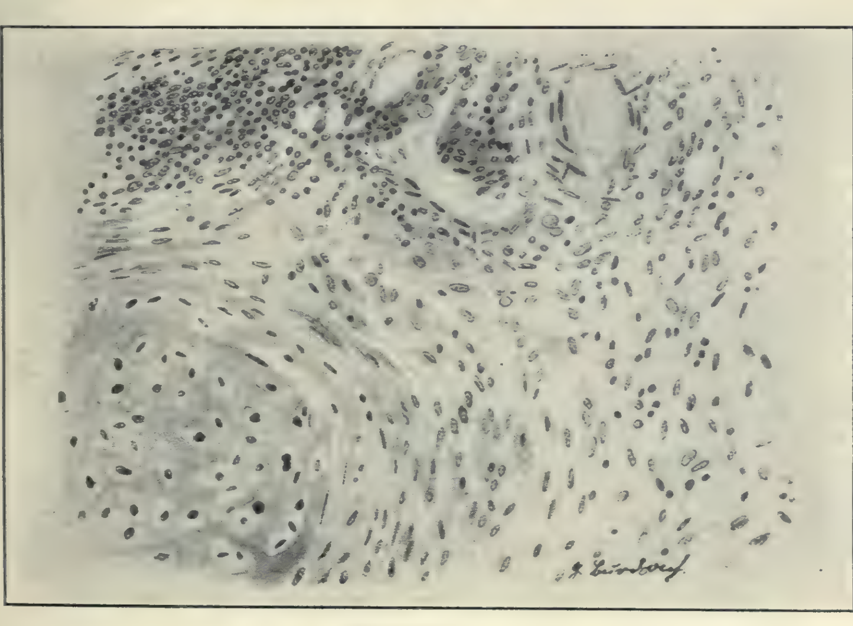

FIGURE 2.

XVIII.

AIR AND RHINITIS.

By Walter J. Wurtz, M. D.,

Buffalo.

In considering rhinitis we often refer to it as "cold in the head." In this reference we cannot confine ourselves to one disease. The term "cold in the head" is vague, but not entirely meaningless. The term was coined many years ago and is here to stay. If we would only think of the meaning of the words "catching cold," we probably would not use them as often as we do. How often have all of us here told our patients that they have "caught cold," when their wound suppurated, or "caught cold" where there was a relapse in some febrile condition, or following an obstetric case. However, it only means one explanation—that is, to satisfy the laity.

In the etiology of "cold in the head," I believe hygiene to be one of the first considerations. Hygiene of the body as well as hygiene of the home, schools and other public buildings must come under our observation. Under hygiene of the body we must look well to our eliminative organs and digestion, keeping the skin active and bowels regular, thereby lessening the chances of an autointoxication. The body should be well protected but not overburdened with clothes, so that radiation is assured. Some authorities advocate the same weight underwear all the year round, and increase the top clothing as to weather conditions. The hygiene of the home and public buildings should be looked after, and is of equal importance. The air should be changed often and proper humidity maintained. The average humidity of a room should be from forty per cent to sixty per cent, but in our climate it cannot be over twenty per cent, due to the extreme cold which lowers the dew point. The humidifying of air and the removal of dust from factories has improved some factories so that the attendance of their employes has been close to one hundred per cent, where formerly it would be from sixty and seventy-five per cent because of the "colds" they "caught" during inclement weather. The subject of ventilation, with its kindred branches, humidity and dust elimination, has become a study which is employing the serious consideration of scientists the world over.

Volumes have been written, large sums expended in experimentation by individuals and corporations, municipalities enact and enforce ordinances for the betterment of conditions, arichtects and engineers strive to plan and construct heating and ventilating plants to give the best possible results, and engineering and medical societies are giving a full share of their time to this work. All these have made great strides in the right direction, but still there remains much to be done.

The ventilation and sanitation of large buildings, such as office buildings, hospitals, schools, churches, theatres and factories, must be considered as to the purity of air and proper humidity. The question naturally arises as to "what is fresh air." From a ventilating standpoint, fresh air is air free from dust and bacteria and a minimum amount of carbon dioxid (CO_2).

Ordinary fresh air, or outdoor air, contains on an average four parts of CO_2 in ten thousand.

Mr. Chas. S. Churchill reports as low as 1.76 to ten thousand in Paris, and 1.75 in London. These are unusually low, and may be due to the method of determination, as the records of the Montsouris Observatory at Paris gave as an average of observations covering thirteen years, 3.14.

Dr. G. A. Soper reports an average content for two thousand observations of New York City air of 3.61 and a maximum of 5.61 and a minimum of 2.69.

CO_2 is harmless, even in the most poorly ventilated rooms. The feeling of depression is usually due to high temperature and high relative humidity.

Dr. E. Vernon Hill, in the report of the ventilating division of the Chicago Health Department, says: "Repeated investigations by physiologic chemists have failed to find any poisonous or harmful ingredients in respired air, but the fact should be emphasized that while such substances have been found, it would be unscientific and illogical to assume that they do not exist. We must, however, discard the belief that CO_2 can be used in any way as a measure of such impurities."

Also CO_2 is a very accurate measure of the amount of fresh air supplied, and by reason of the fact that fresh air bears a direct relation to the bacteria in the air, it is in this way a reliable index to the dust and bacteria.

A room is considered to be fairly well ventilated when the CO_2 is not over six or eight parts in ten thousand.

The amount of fresh air to be supplied per person has been placed at different figures.

Ohio's new building code requires a ten minute air change for churches, lodges, clubs, hospitals or hotels. All other assembly halls and theater auditoriums, not less than twelve hundred cubic feet of air per hour for each person.

Dr. Hill conducted a series of experiments in examining the air collected in public places, also took cultures by exposing Petrie dishes in these places, and examined as to the variety of germs and CO_2. All these showed a great deal of CO_2 and dust in the air.

Further experiments were made where a room had absolutely pure air and an employe was set to work therein. Gradually CO_2 gas was forced in the room, replacing the oxygen. The CO_2 was raised to the point where it was thought no person could exist very long. The man, however, stayed there several hours, and the only thing he noticed was a fullness in the head. He was again placed in the room and dust was forced in; he then developed an acute coryza and could not stay as long as before. This is only to prove that clean bad air has little effect, but the colds we receive are from infected as well as bad air.

Benjamin Franklin in his essay says that "colds result neither from wet nor cold, but from other causes entirely independent of this malady."

Robt. Peary in his diary states that none of his party developed "a cold" in their dash to the North Pole, but they all were attacked a few days after they returned to the settlement. This detail has been gone into only to show the importance of hygiene in "colds in the head."

Dr. Chas. D. Graham-Rodgers, of the New York State Labor Department, recommends for some factories ventilation as high as from three thousand to five thousand cubic feet of air per hour per person.

There are cases in which the amount of air per hour per person is varied to suit the circumstances, a few of which follow:

Hospitals (ordinary)	2100 to 2400
Hospitals (contagious)	4800
Workshops	1500
Prisons	1800
Theaters	1200 to 1800
Meeting halls	1200
Schools (for children)	1200
Schools (for adults)	2400

Fresh air may be supplied in sufficient quantities, but if too dry, disagreeable sensations are experienced. Therefore, it must contain a certain amount of moisture. The amount of moisture which the air will hold depends on the temperature. For instance, at 70° it will hold eight grains per cubic foot, while at 32° it will hold two grains, and at 0° only one-half grain. Since air normally has a humidity of from forty to fifty per cent, air at 70° should contain from three and one-half to five and one-half grains, and at 32° about one and one-fourth grains. But when taking outside air for ventilating in the winter at 32° and heating it to 70°, it would have a humidity of only fifteen and one-half per cent; or if taken in at zero, it would have only from three to five per cent, a condition dryer than that of any desert.

Dr. Henry Mitchell Smith, in an article entitled "Indoor Humidity," says: "The overheating of our houses has been accepted as a prominent cause for catarrh, but I am confident that the low relative humidity and consequently the large saturation deficit of the aqueous vapor in the atmosphere of our rooms is much more important." Also, "It is easier to 'take cold' in a room at 72° with a relative humidity of thirty per cent, than in a room of 65° with a relative humidity of sixty per cent."

Dr. W. A. Evens proposes the following for school ventilating: "First, reduce the temperature of rooms to 68°. In such a temperature the exhaled air, being hot and moist, will rise out of the breathing zone and be replaced by purer air.

Second, raise the relative humidity to sixty per cent or seventy per cent. If the relative humidity is raised to sixty per cent, the pupils will be comfortable with a temperature of 68°. Maintaining a moderate humidity tends to keep the dust inactive and renders persons less subject to draughts. Third, the amount of dust in the air depends on climatic conditions and local influences. There is more dust in a manufacturing district than in a residential section, and less in the country. There is also more dust in the summer than in the winter."

Bruce L. Cushing, to whom I am indebted for the engineering data, recently made some tests on indoor and outdoor air, with the following results:

"In a tight room with a temperature of 70° and a relative humidity of fifty-five per cent (unwashed air, in motion) there was one milligram of dust in every thirty cubic feet of air.

"With the ground covered with snow, an average temperature of 40° and a humidity between forty per cent and fifty-five per cent, there was only one milligram of dust to two hundred cubic feet of air. The latter seemed to be mostly soot or factory smoke."

These were long-time tests, taken with an apparatus which was constructed under Mr. Cushing's supervision, which is, to his knowledge, the only accurate long-time testing instrument ever used.

Dr. Hill places the maximum amount of dust for good ventilation at 4.2 particles per cubic foot, and the number of bacteria colonies on a five minute plate not over twenty.

To insure a supply of clean pure air for ventilation, an air washer is necessary. (An air washer is a metal case containing, in the order named, a distributing plate and one or two banks of spray nozzles and eliminators. Under this case is located a water tank, from which the spray water is pumped to the nozzles and to which it returns.)

There are three primary requirements in the design of an effective air washer and humidifier:

First.—The Spraying System: (a) Must be uniformly distributed to treat all parts of the air currents alike. (b) It must freely atomize the water and thoroughly mix with the air, in order to wet and make heavy the smaller or minute particles of dust and dirt carried by the air. Large drops of spray or sheets of water will not wet these smaller particles. (c) The nozzles should discharge in the direction of air flow, so a minimum resistance is offered to the air current. (d) The spray nozzles should have large, unobstructed orifices, so they will not clog. (e) Proper and adequate filtering must be provided, to prevent dirt and other obstructions from entering the spray piping and nozzles.

Second.—Washing Surface: The removing of dirt, dust, soot and bacteria from the air is almost proportional to which it is exposed. Some of the heavier solid matter is precipitated to the tank by the spray, but the smaller particles, even after wetting, are carried in suspension by the air currents. In order to effectively remove this very fine material, the air must be broken into thin layers or strata and passed over a surface kept flooded with running water. This surface should be arranged to provide changes in the direction of air flow in order to throw the dust particles against it and wash them to the tank.

The Carrier air washing and humidifying apparatus is being installed in many of our modern buildings and schools. A public school so equipped was situated next to a garage. The odor of gasoline was constantly in some of the rooms. After the installation of an air washing and humidifying apparatus there was deposited in the receiving pan a layer of mud, a layer of cylinder oil, and a layer of gasoline, all collected from the air of this school. These washers remove ninety-eight per cent of the dust, dirt and bacteria from the air. Five pails of dirt were washed from the air used for ventilation of Public School No. 6, Brooklyn, New York, in one week. No doubt equally as much more was washed out when the tank was drained. One hundred and twenty-five pounds of dirt, carrying disease, some of which otherwise would have been inhaled by the pupils and the balance have settled on the furniture or been kept in circulation, was certainly worth eliminating.

The varieties of "cold in the head" depends entirely on the diagnosis. The varieties which we meet are: acute, chronic, caseous (inflammatory or suppurative), diphtheritic, and syphilitic. Of the chronic form we have the hypertrophic, atrophic, and polypoid degeneration, acute and chronic sinuitis, foreign body in the nose, etc. Any of the above mentioned conditions produces an effect on olfaction, phonation, and respiration.

The main physiologic function of the turbinate bodies is to warm and moisten as well as to filter the air. The mucosa of the nose has tremendous power for radiation and moistening the air. The estimated amount of secretion in twenty-four hours is supposed to be about one pint. Cold air entering the nose is warmed at body temperature by the time it reaches the larynx. The physiologic function of the accessory sinuses of the nose is probably to promote resonance and also to dissipate shock in case of injury to the cranium. The specific duty the ethmoid cells perform is the nonconducting of heat or cold. The importance of this is recognized when we can see how the delicate structures about the orbit are protected.

In an acute rhinitis the patient's first complaint is dryness of the nose, slight temperature, and probably some headache. This is soon followed by a serous discharge. The mucosa in the beginning is engorged, swollen and dry. Later, following the transudation of serum from the blood vessels, the mucosa is still swollen but bathed in moisture. The discharge soon becomes viscid and involution soon follows.

In chronic rhinitis the patient complains of heavy sensation in the forehead, listlessness, and mucopurulent discharge. The mucosa is swollen and may have the appearance of tumefaction. It is pale, and the discharge adheres to the mucous membrane. On cocainizing, the membrane shrinks tight about the turbinates. This condition is also known as intumescent hypertrophy.

In true hypertrophy the membrane appears pale, discharge is viscid, and on cocainizing the membrane does not shrink about the turbinate bodies.

In all cases of rhinitis it is difficult to say where rhinitis ends and sinuitis begins.

With all the foregoing conditions a sinuitis may have been superimposed. We rarely see a true simple rhinitis without an accompanying sinuitis. In all inflammatory conditions of the nose, consideration must be given to deformities, foreign bodies, and tumors. By tumors we refer to the various polypoid degenerations, fibroid tumors and other neoplasms. Only recently I had occasion to see a patient with a fibroid tumor that filled the entire nasopharynx and extended down close to the epiglottis. The uvula was pushed over to the right side of the mouth, lying against the jaw. This condition causes a degenerating process to go on in the nose due to the mechanic force—pressure.

In diseases of the accessory sinuses of the nose that give rise to various forms of rhinitis, we must consider the maxillary antrum, ethmoid cells, frontal sinuses, and sphenoid cells. In diseases of the maxillary antrum, proper drainage is the thing to accomplish. If polypoid degeneration has taken place, the antrum must be opened radically and all polypoid degenerating tissue removed. In ethmoid disease always be sure of the diagnosis, for the physiology of the cells must be kept in mind. As nonconductors of heat or cold, their protection of the orbit is necessary, therefore, complete exenteration should be done only when infection is beyond medical treatment.

Early in ethmoid disease stimulating treatment will produce marvelous results. In complete destruction of the ethmoid cells the middle turbinate is always sacrificed. The sense of smell is sometimes impaired by too radical an operation. Often, however, the sense of smell has been destroyed by preceding rhinitis.

In frontal and sphenoidal sinus disease, drainage again is the important factor. In operating on the sinuses one must always be prepared for various malformations, as no two sinuses are alike. In all long-standing discharges of the nose, where the discharge has been profuse, we often see hypertrophy of the nose and upper lip, with excoriation of the skin of those parts.

There is also a condition described by Vaughn in which the mucous membrane assumes a pale appearance and very much swollen. This often follows certain attacks of asthma and hay fever. He attributes this to protein poison in the circulation, producing a toxin affecting these parts. If we can get more information as to the character of these toxins we may soon have a remedy for hay fever and certain cases of asthma.

In conclusion, I wish to emphasize the importance of making a differential diagnosis when we see a case of "cold in the head," and also the importance of hygiene in its relation to the onset and cure of this malady.

I am indebted to Mr. Bruce L. Cushing, C. E., for the accurate data pertaining to the engineering portion of my paper. He is associated with Mr. Carrier, the inventor of the air washer and humidifying apparatus.

XIX.

INTUBATION.*

By U. S. Bird, M. D.,

Tampa.

Intubation, in the treatment of infantile laryngeal diphtheria, stands alone as the most dramatically effective procedure in medicine. The change wrought by the tube is amazing, especially to the novice. It was formerly unique in its usefulness, but that has been abridged by the advent of a method without a superior in medical practice. A large factor in the usefulness of antitoxin is its freedom from the spectacular details incident to intubation, an operation requiring some degree of trained skill, while the serum, if necessary, may be given by a layman. At first thought it is almost regretted that so successful a procedure should have been so entirely eclipsed in its zenith and relegated to the department of rare operations; but a second thought accepts the situation with a profound appreciation of the beneficent efficiency of the serum. While we may expect improved methods of treatment and education to progressively limit intubation, it will not disappear entirely. There will always be a small but certain number of severe or neglected cases requiring instrumentation. Following is a brief record of my cases:

Case 1.—In November, 1907, I was called by Dr. O. about noon to see a dyspneic boy, about two years old, with diphtheria. The case was not urgent, and we kept it under observation till midnight, when a tube was placed on the third attempt, with immediate relief. Next day the tube was expelled and was replaced by a larger size, on the first attempt, an hour later. This was coughed up three days later, without unpleasant result, recovery proceeding uninterruptedly.

Case 2.—In March, 1908, I was called by Dr. B. to see a

***Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society.**

case with the usual history. The boy was four years old and in poor general condition. A tube was placed on the third attempt, the case proceeding without incident. This case terminated unfortunately. The tube was removed at the second attempt, on the fourth day, the larynx being and continuing in good condition; but death occurred within a week from gastroenteritis.

Case 3.—Seen with Dr. B., May, 1910. This case, a boy of two years, was uneventful, excepting the laryngeal emergency. The tube was placed on the third attempt, and removed on the first attempt on the fifth day, recovery being without incident.

Case 4.—This was a serious case, seen December, 1912, with Dr. R. The boy, four years old, was exhausted and languid, respirations shallow and difficult. Three attempts were necessary. When I was satisfied that the tube was in place, respiration had ceased, and he was cyanosed and limp. He was hastily placed on a table with head over the edge, and artificial respiration used for a minute before it was responded to. Recovery proceeded rapidly, and in twenty minutes he was sitting up and taking milk. This was the only case in which I left the cord in place, attached to the cheek by adhesive plaster. The tube was removed five days later, to be replaced two hours later, at the third attempt, again leaving the cord. Five days later it was removed permanently.

Case 5.—This was the most troublesome of my cases. I was called by Dr. B. to see a girl of two years, December 20, 1912. It was a neglected case of diphtheria with labored respiration. The tube was placed on the third attempt, with rather delayed relief. On the sixth day the tube was removed on the second attempt, being replaced two hours later, on the first attempt. On this occasion the doctor was absent, and I placed the tube with the assistance of members of the family, with little trouble. On this and subsequent instrumentations of this case, one attempt sufficed. Six days later it was removed on the first attempt. In the light of the previous experience I anticipated about the same interval in case replacement should be necessary. I hurried back an hour and a half later, in response to repeated calls by the family and the doctor, finding the baby in much distress, which was relieved by a hurried intubation. Three days later the tube was expelled and a larger size used, which was retained two days, and replaced by a still larger size. This was expelled after four days, but the condition remained satisfactory thereafter. On this case I did five intubations and three extractions. I see the patient occasionally. The voice is rough, but has improved lately.

Case 6.—The treatment in this case was brief. In March, 1914, I was called by Dr. F. in the forenoon to see a diphtheritic, dyspneic boy of three years. A tube was placed on the second attempt, to be expelled an hour later, and replaced by a larger tube on the first attempt, which was expelled on the following day. The condition being satisfactory, the case was left with the attending physician.

Case 7.—This was a case according to the rule. In April, 1914, I was called by Dr. S. to see a boy of three years, with the usual history and condition. A tube was placed at the third attempt, left in five days, and removed at the first attempt without subsequent incident.

Case 8.—This was another case giving little trouble. I was called by Dr. I. on a morning in last June, to see a boy of two years. A tube was placed on the first attempt, to be expelled an hour later, and be replaced by a larger size, on first attempt, which was expelled three days later without further incident.

My experience in this small series of cases has developed nothing out of the ordinary. There were no deaths from the condition treated. The only one in the series was Case 2, from a trouble not related to diphtheria. The eight cases represent sixteen intubations and six extractions. In six cases three attempts were necessary for the initial intubation; in one, two attempts; and in one, one attempt. In the four cases in which one additional intubation was done, one required three attempts; the other three, one each. In the case requiring four additional intubations, one attempt each sufficed. I had little trouble with my extractions, one or two attempts sufficing for each.

The technic is well known. The guides are the epiglottis and the arytenoids. Sometimes the latter only can be distinguished; the epiglottis imparting a mushy sensation to the finger, indicative of nothing in particular. The Feroud instrument is the only one satisfactory to me. I have bent the obturator and removed the tube, after having placed it, in removing the O'Dwyer intubator. Although I have been prepared for tracheotomy on occasions of intubation, my cases have developed no condition requiring it, and I anticipate no such trouble. If membrane should be pushed down and stop the tube, its prompt removal by the cord may be expected to relieve the emergency. Contrary to cautions lately appearing regarding the use of too large tubes, my trouble has been to get a tube large enough to stay in. In one case I twice increased the size of the tube, and the last was finally coughed up. I would not consider replacing the same tube, unless in a hospital where help was always at hand. Even then it would be a waste of time. All of my cases were at their homes. I have had little trouble with nourishment. Nursing has always been possible. I have given nothing but liquids, and they were usually taken from a spoon, in any posture. Careful preparation is desirable, but these cases are emergencies, and it is surprising how little preparation and assistance suffice. Sterile instruments are necessary, and a gown and gloves, also sterile, are a comfort, but on occasion formalities may be dispensed with without risk to the patient.

Two reliable persons, one to sit and hold the child, the other to hold the gag and head, are usfficient. On one occasion my preparation consisted in removing my coat, rolling up my sleeves, rolling the child in a sheet, placing it in the lap of some one in a straight chair, adjusting the gag and getting some one else to hold it and the head. Within three minutes after I entered the house the tube was in place, and I indulged in the luxury of a long breath; my removal of the tube two hours before was the cause of the emergency.

Since writing the above two other cases have come under my care.

Case 9.—On November 8, 1914, I was called by Dr. B. to see G., a girl, eight years old, the oldest of my cases. She was in distress, having been kept awake some thirty-six hours by respiratory efforts. A tube was placed on the third attempt, with immediate relief. On the night of the 11th the expulsion of the tube was uneventful, recovery proceeding without further incident.

Case 10.—Seen with Dr. S., November 17, 1914, a boy, two years old, thought to be in extremis. The tube was placed on first attempt, within five minutes after arrival, with prompt relief. In this case the diagnosis was not made till after intubation, the laboratory finding at first being reported as "suspicious." On the 23rd the tube was removed on second attempt at 8 a. m., to be replaced at 1 p. m., first attempt. On the 27th, removed at 8 a. m., replaced an hour later, one attempt sufficient for each procedure. December 2nd removed tube on third attempt, replaced an hour later on second attempt. During the manipulations on this occasion I recognized the probable cause of failure in my abortive efforts, which may also explain other failures. While the left index finger is holding up the epiglottis and palpating the laryngeal introitus, it monopolizes the operative field, and must be withdrawn sufficiently to give access to the laryngeal entrance, or further successful manipulation is prevented. But in this withdrawal care must be had not to release the epiglottis. That is what I seemed to have done, with resulting falling of the epiglottis, closure of the entrance, and passage of the tube into the esophagus. With the epiglottis down, no effort should avail to effect an entrance. Bearing this in mind, the next instrumentation in this case, an extraction, was without difficulty. On the 7th the tube was removed on first attempt at 8 a. m. For an hour the result seemed doubtful, but a chance was taken on leaving it out, and the case proceeded without further interference.

XX.

COMPLICATIONS IN TONSIL AND ADENOID WORK, WITH ESPECIAL REFERENCE TO CHRONIC SUPPURATIVE OTITIS MEDIA.*

By Harvey Mayer Becker, M. D.,

Sunbury, Penn.

The title of my paper, as it appears upon the program, is somewhat misleading, as it would indicate that I expected to deal with complications arising during tonsiloadenoidectomies, which is by no means my intention, and I trust you will pardon the attempt to discuss one complication present before operation.

I have a feeling of considerable hesitancy in appearing before you with this modest paper, and I hope you may not think that it is with any presumptuous idea of teaching you anything new, but rather that we may rehearse together some of those conditions which I desire to call complications in tonsil and adenoid work. I prefer to call them complications rather than sequelæ, inasmuch as they frequently antedate the attention to the tonsil and adenoid. We are frequently consulted for the purpose of treating the complications, and the cure of the complications depends upon the active and judicious management of the actual cause, of which the patient and his family are wholly ignorant, or have been grievously misinformed.

It will be obvious to you that it would be utterly impossible for me to take up for discussion more than one of the very many complications in the time allotted; nor would I feel myself justified or qualified in making the attempt. It should not be amiss, however, to name over some of them, just to refresh our minds and make us realize that there are other parts of the human body that should interest the laryngologist besides the much censured and abused tonsil, which we should well consider, in such a far-reaching combination of afflictions, before we proceed with our treatment, and which should be our aim and guide in determining the proper method of procedure.

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*Read at the meeting of the Philadelphia Laryngological Association, January 4, 1916.

We may say that a lymphadenitis of the globe and orbit is not rare, that we may have rhinitis, pharyngitis, laryngitis, accessory sinus disease, bronchitis, swollen and suppurating glands in the neck, obstruction to breathing, short lip, lack of development of the superior maxillæ, high palatal arch, deviated septum, irregular spacing and malposition of the teeth, which at once leads us into the comparatively new but most important field of orthodontia, defective speech, tuberculosis, nephritis, endo- and pericarditis, septic arthritis and metastatic abscesses, otitis, mastoiditis, perisinuitis, sinus thrombosis, brain abscess, and so on. These, then, are some of the conditions frequently present as a complicating factor in our tonsil and adenoid work; but there is only one which shall be considered at this time—otitis media. The mention of any complications during or subsequent to operative treatment has been intentionally omitted.

In the consideration of otitis, no attempt shall be made to describe the anatomy of the parts involved, except insofar as the anatomic structures may have a bearing upon the mechanical cause. It is quite certain that otitis, whether of a mild chronic variety, leading to tinnitus, otosclerosis and loss of hearing, or of a severer type, leading to an acute or chronic suppurative process, is produced by several modes, which may be said to be mechanical, including obstruction, inflammatory and bacterial. Several modes of mechanical cause may exhibit, as, for instance, direct obstruction by adenoid tissue in Rosenmüller's fossæ, or packed directly about the funnel-shaped orifice of the eustachian tube. Small adenoid vegetations so placed are capable of causing more harm to the ear than larger masses centrally located in the epipharyngeal space. Just what the exact relation of the pharyngeal musculature is to the ventilating processes of the ear is perhaps not any too well understood, but certain it is that the palatopharyngeus muscle extends to the pharyngeal orifice of the tube, so that inflammatory states in the closely associated tonsil, and necessarily in the pillars, may readily extend along the pharyngopalatin folds to the mucosa of the tube and thence to the middle ear. Persistent chronic and oft-recurring inflammatory conditions in the tonsil, and secondarily in the pillar and pharyngeal muscles, will greatly impair their function, resulting in the impairment of the muscular mechanism controlling the patency of the tube. Normally the tubal epithelium is ciliated, the movements of which cilia are toward the epipharynx, for reasons which are perfectly clear. From repeated inflammatory attacks in the tube this function is impaired or lost, and consequently when infecting material, laden with a variety of pathogenic bacteria, is forced into the tube by sneezing, coughing, blowing the nose, or by any of the numerous modes, the function of the ciliated epithelium being greatly inhibited or lost, these bacteria have an unimpeded passage to the middle ear cavity—a through ticket, so to speak—with rarely an attached return coupon. Once having gained access to the cavity, suitable media and conditions for prolific propagation having been found, it depends only on whether sufficient resistance to the invasion is present, whether or not a suppurative process is created.

A study of the bacteriology of suppurative otitis soon becomes convincing of the fact that a number of different germs are capable of producing this disease, the variety depending entirely upon the variety of germ present in the epipharynx at the time infection took place. Probably the most common is staphylococcic, which is followed by streptococcic as a close second. Pneumococci, the germ of influenza, whatever this may be, micrococci catarrhales, colon bacilli, and undoubtedly the organisms of scarlet fever and measles, which unfortunately have not been isolated with a certainty, but perhaps are closely allied to streptococci, are capable, either in pure culture or in mixed infections, of causing most intractable suppurations. Bacteriology has conclusively shown that any one of these or all of them may be present in the crypts, and even in the stroma of the tonsil which is diseased, from which they are easily distributed to the entire oral and nasal cavity. It is not the intention of the author to enter into a discussion as to whether they were bred there or whether they are being held there until their destruction could be accomplished—two much disputed theories at the present time, neither, perhaps, any too well founded on fact—but their presence there in large numbers, in increasing numbers, should be sufficient evidence to convict their host and shelterer as an accessory before the fact.

A diseased tonsil, therefore, whether enlarged or not, necessarily stands convicted of being capable of causing numerous otitic suppurations, which I believe to be incurable so long as this offending tonsil is permitted to remain in its fossa, and for this reason I begin my treatment of the otitis by a thorough cleaning of tonsillar fossa and epipharynx. The extracapsular operation in children, and the intracapsular in adults, are my choice. In adults it is frequently not possible to remove the capsule without injury to the pharyngeal aponeurosis, which is usually attached and adherent to the capsule from chronic inflammatory processes, and without exposure of the underlying musculature. I feel that it is wiser to confine the operation within the capsule, under these circumstances.

In this unhesitating complete removal of the tonsil I am not unmindful of its much disputed function, and most highly appreciate the endeavors of the many observers and investigators, the earnest pleas of such men as French and Jervey, for conservatism, and the untiring efforts of our esteemed G. Hudson Makuen in his constant endeavor to instill into our minds the fact that the unaltered throat and perfect voice are also important factors in the life and welfare of the individual, and a lack of quotation from their writings I would have construed as being due to limited time and not a failure in appreciation.

Certain it is, however, that if the offending tonsil has the marked effect upon the growth, hearing and mental development of children as just recently so well stated by D. Braden Kyle, from which there is no retraction, and certain it is, also, that when it has become sufficiently diseased to be the great factor in otitis, it has necessarily ceased functionating, just as any other organ does when severely diseased, and must be considered a menace, and of no further use to the patient.

Neither am I unmindful of the teaching that at the time of the tonsil and adenoid operation the ear had better be left alone, and subsequently treated on general lines for chronic suppurative otitis media. I shall say, at this point, that a tonsil operation should never be done during an attack of acute otitis.

I do not believe much can be gained in any discussion by the citation of individual cases, and shall not do so, but wish to present a series of twenty-two patients, with thirty-one discharging ears, discharging from two months to two years, in which treatment was given at the time of the tonsil and adenoid operation. These ears showed various germ infections, mostly staphylococcic and streptococcic, several with mixed infections, one of colon bacillus, and two postscarlatinal. Thirty-one ears are not a sufficient number to put the method upon a sound basis, but the results have been so universally satisfactory that I desire to present them to you.

Reasoning from the fact that infecting materials are more easily driven into the tube when perforation of the tympanum exists, the method appears to be contrary to the rule. I wish to say that the treatment has not been tried in acute suppurative otitis media without perforation, the tonsil operation having been deferred until the otitis has been cured, either by primary incision or spontaneously, when the ear probably will not need any further attention. To the present time it has been tried only on the chronic case with perforation present, and is based upon the principle that the whole air conducting mechanism of the ear has been infected, from the pharyngeal orifice of the eustachian tube to the meatus of the external auditory canal, and must be wholly asepticized at the same time.

METHOD.

Immediately after the tonsil operation the ear canal is wiped free of all discharge and the condition of the tympanum and middle ear cavity carefully determined as far as possible. If, as sometimes happens, the greater part of the tympanum has been lost, access to the tympanic end of the tube is considered sufficiently well established, but if only a small perforation is present, a long posterior incision is made, with an attempt to include the perforation in the incision. If this is not possible, owing to anterior location of the perforation, it is disregarded. After gentle aspiration with a Siegle otoscope, the canal is again dried. A five per cent alcoholic solution of iodin is freely placed into the canal, permitting it to flow through the perforation or incision into the middle ear cavity; and it is surprising with what ease it apparently finds its way down the eustachian tube, by the favorable position of the head, not being retarded by air pressure. It may be found necessary to gently use the otoscope, alternately making pressure and suction. After a few moments a dry gauze pack is placed in direct contact with the drum, not too tightly, but sufficiently snug to prevent the passage of air upward through the tube and out through the canal. In about forty-eight hours the pack is removed.

Of the thirty-one ears thus operated, only four had not ceased to discharge at the time the pack was removed. One of these ceased after six days. The remaining three were the one colon bacillus and two scarlatinal. The colon bacillus ear ceased after four weeks; one scarlet fever after two months, while the other scarlet fever has not ceased after one and a half years, having been subject to several exacerbations during this time. In some of these cases granulation tissue was present in the middle ear cavity, which did not seem to retard the cessation of the otorrhea, but added considerably to the difficulty in properly filling the cavity with the solution. Small perforations closed very kindly under the stimulating effect of the iodin and pack, as did also the incisions. Several large perforations required some stimulating treatment afterwards, while a few very large ones I have not been able to close, even in the presence of what appears to be a dry ear. In the scarlet fever ear still discharging, the greater part of the tympanum was lost, with necrosis of the ossicles, and some more radical procedure will be necessary for this ear.

Details of the hearing present in these ears before and after operating would consume too much time, and for the sake of brevity these are omitted. As a class of suppurating ears in which usually considerable hearing had been lost, the great majority showed improvement that was very gratifying, while a few were not improved, notably one ear in which otorrhea had been present for over two years. In no ear was there less hearing after operating than before, another gratifying feature, in view of the fact that with the persistence of the otorrhea further loss would necessarily have taken place.

Naturally, one would inquire if the control of the otorrhea is to be permanent. The usual difficulty of following cases is to be expected, but the explicit instruction to the parent, and so far as possible to the family physician, to return the child for inspection once a month, or at the first sign of any trouble with the ear, has usually met with success, and a fairly close watch has been kept over these cases, and none have been returned uncured except as above stated.

XXI.

THE IMPORTANCE OF THE VESTIBULAR MECHANISM IN NEUROLOGY.*

By J. Ramsay Hunt, M. D.,

New York.

The otic labyrinth is of great interest and important neurologically, as it is the terminal end organ of an extensive sensory mechanism which has connections with many portions of the central nervous system. This mechanism is frequently involved in affections of the nervous system—organic, functional and psychic—and when the seat of pathologic disturbances, produces symptoms which are essentially neurologic in character—e. g., vertigo—subjective and objective; disturbances of static consciousness and equilibrium; nystagmus, and forced movements due to an alteration of the labyrinthine tonus of the muscles of the trunk and extremities. A knowledge of the functions and relations of the vestibular nerve is, therefore, essential to a proper understanding of vertigo, equilibrium and orientation, nystagmus, and cerebellar localization.

ANATOMIC CONSIDERATIONS.

The otic labyrinth may be regarded as a highly specialized end organ of the vestibular division of the eighth cranial nerve. As is well known, the auditory nerve consists of two groups of fibers which are functionally quite separate and distinct—viz., the cochlear, which is the essential nerve of hearing; and the vestibular division, which is concerned with the important function of equilibrium and the space sense. These two groups of fibers course in a common trunk, the acoustic or eighth cranial nerve. Their peripheral and central terminations are, however, quite distinct and separate. In this discussion we are concerned only with the vestibular nerve and its relation to static consciousness and equilibrium. The peripheral stimuli which carry sensations of movements of the head take their origin from wave-like movements of the endolymph within the semicircular canals. The fluctuations in movement of these fluid columns are registered by waves of the delicate cilia on the epithelial cells of the cristæ ampullarum; from here the impulse is carried to the ganglion of Scarpa, a small peripheral ganglionic structure of the vestibular nerve and the homologue of the posterior spinal ganglia. From here the sensory impressions flow along the fibers of the vestibular nerve to the primary nuclei in the medulla oblongata. Of these there are several aggregations, of which the more important are the nucleus of Deiter and the nucleus triangularis; other accessory ganglia are the nucleus of Bechterew, nucleus of Staderini, and the nucleus vestibularis descendens. From these primary nuclei the labyrinthine impulses are diffused in many directions, viz.:

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*Read before the Section on Otology, New York Academy of Medicine, December 5, 1915.

1. To the ocular nuclei on both sides, by way of the posterior longitudinal bundle. Through this mechanism vestibular nystagmus is produced.

2. To and from the vermis and cerebellar cortex, as well as other ganglionic structures in the cerebellum.

3. To the anterior horn cells of the spinal cord through the vestibulospinal tract. (The connections of two and three produce the forced movements, disequilibration and the alterations of muscle tonus of labyrinthin origin.)

4. To the region of the nuclear origin of the vagus nerve, which explains the nausea, vomiting, dyspnea, and circulatory changes of severe labyrinthine vertigo, and finally:

5. Fibers which pass cephalad through the midbrain and the optic thalamus to the cerebral cortex, terminating, it is supposed, in the region of the posterior portion of the parietal lobe. This communication with the cerebral cortex is the anatomic basis for the existence of static consciousness and affords an explanation for the disorientation and other psychic reactions of a severe vertiginous seizure.

Of great practical importance at the present time are the afferent and efferent communications between the primary vestibular centers in the medulla and the cerebellar cortex, which were demonstrated by Ramon y Cajal by the silver impregnation method. Cajal has also shown that collaterals pass from the pyramidal tract fibers in the pons varolii to the cerebellar cortex of the opposite side. Therefore, the motor fibers of the pyramidal tracts stand in relation to the cerebellar cortex, and the labyrinth has extensive connections with the cerebellum and the spinal cord. These communications explain in part the occurrence of ataxia, forced movements, and alterations in muscle tonus which are observed in labyrinthine disturbances. They are also of especial importance in reaching a proper understanding of the reaction movements of the head, trunk, and extremities, as described by Bárány after the caloric test and rotation experiments.

This, then, represents, roughly, the course and terminations of the vestibular nerve, and is, so to speak, the anatomic basis of vertigo. It is a bilateral mechanism, and for its proper function a strictly harmonious action is required from each side simultaneously. If there is irritation or paralysis of the end organ on one side an asymmetric stimulation results, with the production of true labyrinthine vertigo, viz., sudden disturbance of equilibrium, nystagmus, a sense of subjective and objective rotation of objects, severe vertigo, nausea and vomiting, pallor, tachycardia, dyspnea, darkening of the field of vision, and sometimes unconsciousness.

LABYRINTHINE NYSTAGMUS.

The nystagmus of vestibular origin is rhythmic in character and consists of two components, a slow movement followed by a rapid return movement. The direction of the nystagmus —i. e., to the right or left, upward or downward—always refers to the direction of the rapid movement. It is always more manifest when the eyes are turned in the direction of the rapid nystagmus; it is diminished or suppressed in the direction of the slower component. The slow component is the direct result of the vestibular irritation, while the rapid return movement is of cerebral origin. Pike has shown, as a result of experimental study, that the rapid phase of the nystagmus is probably produced from the contralateral cerebral cortex. It is of especial interest to note that each semicircular canal evokes ocular movements in its respective plane.

Bárány has perfected an elaborate series of tests by caloric and rotation stimulation of the various semicircular canals, which are of great interest physiologically and of great practical importance. These tests not only determine the functional activity of the vestibular nerve, but a proper understanding of their detail and technic is essential for the interpretation of the various disturbances of labyrinthine tonus. Nystagmus of labyrinthine type is also produced by lesions of the vestibular nerve and of the central connections in the medulla. There is some doubt as to the relationship of cerebellar nystagmus to vestibular nystagmus. Wilson and Pike believe that pure cerebellar nystagmus is essentially an ataxic disturbance, an incoordination or asynergia of the ocular mechanism, and identical with the asynergic disturbances of cerebellar origin. While this may be true, it must not be forgotten that clinically most cerebellar lesions exert pressure on the underlying brain stem, and in this way may produce true vestibular nystagmus. It would also seem probable that irritation of the vestibulo-cerebellar fibers might have a similar effect.

MOTOR REACTIONS AFTER LABYRINTHINE STIMULATION.

That the labyrinth exerts a tonic influence on the voluntary muscular system was clearly shown in the epoch-making experiments of Ewald. The labyrinthine tonus is exercised through the connections existing between the vestibular mechanism, the cerebellum, and the spinal cord. It is homolateral —i. e., each hemisphere of the cerebellum stands in relation to the corresponding trunk and extremities. The movements of the body take place in the plane of the nystagmus and in a direction opposite to the rapid nystagmus—e. g., if with the head in the upright position and the right arm extended there is induced a horizontal nystagmus to the right, the arm deviates slowly to the left. Bárány has elaborated with great detail and exactness a series of tests for the determination of this tonus mechanism of the labyrinth and its influence on the movements of the extremities and trunk. This mechanism is homolateral in its influence—i. e., the right labyrinth and right cerebellum control the corresponding half of the body. As a result of these observations, he has reached the important conclusion that in the cerebellar cortex the various segments of the extremities are represented by four centers which are concerned with the direction of movements—viz., horizontally (right and left) and vertically (up and down.) A stimulation of the labyrinth on one side induces a corresponding alteration of tonic innervation and the direction of movement. As a result there is produced in certain tests, what he terms pointing by. (Vorbeizeigen.) In other words, the sense of direction of movements in the trunk and the extremities is to some extent under the influence of labyrinthine stimuli, and the induction of nystagmus (rapid movement) in a certain direction produces a deviation of movement in the opposite direction. A very important phase of this question is its relation to cerebellar localization and the practical demonstration of cerebellar disease. It also supplements the experimental work on cerebellar localization of such observers as Rothman, Bork, and van Rynberk; and Bárány has shown that investigation along this line may contribute to a knowledge of the localization of centers in the cortex. For example, by freezing with ethyl chlorid, a portion of the cerebellar cortex which had been exposed by an operation, he was able to show that the postero-inferior surface of the cerebellum controlled the inward movements of the upper extremity.

VERTIGO.

It is important to recognize that vertigo, giddiness, and similar sensations are due to a disturbance of this large neural mechanism, and that clinically we should always think of vertigo in this concrete neurologic sense, just as in visual disturbances one thinks naturally of the peripheral organ of vision and its central connections. If this is done, much that is vague and uncertain in our clinical conception of vertigo will disappear. For example, in congenital affections of the vestibular apparatus, as in some types of deafmutism, the space sense is lost. These individuals may be rotated with impunity and yet experience no vertiginous sensation. What more practical demonstration could be offered to show the dependence of vertigo upon this mechanism?

Vertigo may result from labyrinthine disturbances, disease or injury of the vestibular nerve in the internal auditory canal and at the base of the brain, and affections of the central nervous system, more especially of the brain stem and cerebellum. Organic brain disease is one of the most important and most frequent causes of vertigo—e. g., tumor, abscess, multiple sclerosis, encephalitis, and the like. These may act directly on the nuclear and tract connections of the vestibular system, but frequently the vertigo is produced as a result of secondary circulatory changes and alterations in the pressure of the cerebrospinal fluid.

The vertigo in functional conditions should also be referred to this neural mechanism; so that toxic, gastrointestinal, and neurasthenic vertigo represent limited functional disturbances, toxic or circulatory in nature, from sudden fluctuation in the vascularization of the vestibular mechanism. The not infrequent vertigo of cerebral arteriosclerosis probably has a similar orgin. As we have seen, the ocular mechanism is in close anatomic relationship with the primary vestibular nuclei in the medulla, so that ocular vertigo, especially in association with visual defects and diplopia, is relatively frequent. Ocular vertigo, however, is not purely vestibular, as vision itself is an important factor in static equilibrium and orientation.

A word in respect to psychic vertigo. We have seen that a distressing impression is one of the important accompaniments of a vertiginous seizure. It is an acute painful consciousness of disorientation, produced by disturbances of the vestibular apparatus, a disturbance of the judgment of the relation of body to space, a sensory deception as to posture, direction and distance—in brief, an hallucination of the body sense in its relation to space. We have all experienced vertigo in some of its forms, and retain a more or less vivid memory of the psychic impression; so that, especially in neurotics and in neurasthenic and hysteroneurasthenic states, psychic vertigo may become a dominating symptom. This may persist long after vertiginous seizures have ceased, as a phobia or obsession, and is a pure psychic disturbance, requiring psychic treatment for its relief. A rare and very severe form of this variety is that known as continuous vertigo, the vertigo permanens of Weir-Mitchell. Oppenheim has also described similar states as "Dauer Schwindel." This form of vertigo is clearly allied to the minor psychoses.

We should always bear in mind then that the higher cortical associations play an important rôle in vertigo, which in organic vertigo is the highly unpleasant realization of what is taking place, and in certain functional and psychic states is largely a mental image with associated fear and anxiety.

The treatment of vertigo in general need not be considered in a discussion of this sort. I would, however, emphasize the good results which have occasionally been obtained by lumbar puncture in cases of labyrinthine vertigo. This method was first suggested by Babinski, and Putnam and Blake in this country have reported favorable results. Bárány also recommends it; and from personal experience I can vouch for its occasional efficacy. From five to ten or even twenty cubic centimeters of cerebrospinal fluid are removed by lumbar puncture, with the usual precautions. It may be necessary to repeat the procedure once or twice at intervals of one or two weeks. The exact mechanism by which the favorable result is produced is still somewhat obscure. Probably, however, the changes in intracranial pressure, either circulatory or of the cerebrospinal fluid, is an important factor. It is also not impossible that the tension of the fluid in the semicircular canals may be relieved to some extent by this procedure.

XXII.

THE ROLE OF THE EYES IN EQUILIBRATION AND ORIENTATION.*

BY ARNOLD KNAPP, M. D.,

NEW YORK.

Among the nervous impulses which act in preserving our equilibrium, those obtained from the eyes are mentioned as important. According to Gowers, the knowledge of the relation of external objects to the body gained from the contraction (i. e., innervation) of the eyeball muscles is one of the most important sources of guidance to the centers that regulate the muscular contractions for maintaining bodily equilibrium. Another important accessory to our sense of equilibrium is the static labyrinth. There is a close association between the eyes and the labyrinth. This association, however, is not directly connected with equilibration, but has a different purpose, as shown in the following:

The posterior longitudinal muscle which connects the various nuclei of the ocular muscles is in close relationship with the vestibular nerve. The vestibular nerve thereby is closely associated with the ocular muscles, just as it is with all the muscles of the body. It serves the purpose of maintaining the muscles in a state of tonic contraction. If this nerve be divided on one side, it will result in a complete loss of tone of the muscles of the body of the opposite side and in nystagmus. The vestibular apparatus is also in close relationship with the cerebellum, by means of Deiter's nucleus, so that the cerebellum is a further active factor in the regulation of our ocular movements. Bing calls it a reflex apparatus serving for the preservation of equilibrium, not only for the muscles of the body, but for the eyes. It receives centripetal impulses from the static labyrinth and from the ocular muscles by means of Deiter's nucleus, which assist in its regulating activity. On the other hand, it sends forth centrifugal impulses through this same nucleus, which preserve the position of equilibrium and control the ocular movements.

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*Read before the Section of Otology, New York Academy of Medicine, December 16, 1915.

In certain animals the position of the eyes and their movements are governed nearly exclusively by the ear. The eyes in these animals, if the head is not moved, are practically motionless. As the head moves, the eyes make a compensatory movement in the opposite direction, which originates in the labyrinth. There is a constant action of the labyrinth on the ocular muscles. It holds the ocular muscles in check, giving them a definite tone, so that a lesion of one labyrinth causes a preponderance of the action of the opposite one. In man this association between the eyes and the labyrinth also works for producing compensatory movements, though these are not as important as in the lower animals. The purposes of these compensatory movements, according to Sherrington, are presumably for maintaining the normal relation of the eyes to the horizontal. These movements, Wilson and Pike have shown, are a direct reflex response, independent of the cerebellum, to insure that the retinal images shall not be disturbed by each motion of the head. It is well known that if both labyrinths are destroyed these compensatory movements are abolished.

The chief symptoms, so far as the eyes are concerned, which results from a lesion of the labyrinth, is nystagmus. Nystagmus, however, is a symptom which does not disturb our sense of equilibrium. In other words, those suffering from nystagmus do not complain of vertigo, unless of course, there are other agencies which affect the center of equilibrium.

Ocular vertigo is a very unusual symptom and is difficult to explain. Vertigo occurs, unquestionably, in ocular paralysis, owing to the confusion resulting from false projection; this, however, is a symptom which is surprisingly frequently absent. Muscular anomalies, such as insufficiencies of the ocular muscles, are practically never accountable for vertigo. It is stated by some that certain errors of refraction are apt to cause vertigo, particularly in those who are suffering from astigmatism with oblique axes. The term dizziness, as used by patients that consult an ophthalmologist, embraces many conditions and symptoms which have but little to do with vertigo; it is often impossible to analyze these symptoms or to explain their relation to an ocular defect, particularly when other factors which cause vertigo cannot be excluded.

XXIII.

A SUGGESTION REGARDING THE RINNE TEST.*

By Robert Sonnenschein, M. D.,

Chicago.

While various constructions have been placed upon, and different deductions drawn from, the test devised in 1855 by Dr. A. Rinne, we probably all agree with Bezold that of the tests usually made, namely, the Schwabach, Weber and Rinné, the latter is the most reliable. This is so because in applying it one can more easily get from the patient intelligent and distinct answers, thus noting more definite findings.

Rinne himself performed the test by placing the vibrating fork on the inner incisor teeth, but the method afterwards used by Bezold, Politzer, Urbantschitsch, and the other authorities (such as Boenninghaus, Gruber, Jacobson, etc.), is the one now in vogue, and probably used by all of us. This consists in striking the fork, the one advised by Bezold being a^1 (435 v. d.), and placing the stem on the mastoid process. When no longer heard, the prongs of the fork are held near the auditory meatus and the length of time noted by which the air conduction exceeds the bone conduction, when the test is positive. Where the Rinne proves to be negative, the air conduction is first determined and the fork then placed on the mastoid. Please pardon this trite statement in the presence of otologists, as it is made only for the sake of completeness.

It is not my intention now to discuss the various types of Rinne reaction, their clinical significance, nor their relation to the other functional tests, but will leave such consideration for another occasion. At present I desire merely to point out a fact, which, so far as my perusal of the literature goes, seems to have escaped the attention of most men of late, and that

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is the fact that the air conduction, as compared with that of bone, is really much greater than ordinarily assumed.

Bezold, in his textbook, considers that on the average the air conduction is about thirty seconds longer than bone in the normal positive Rinne, if the a^1 fork is used. However, in one of his great papers on the functional testing of the ear, he states that when the fork is held only at the meatus, without having been previously placed on the mastoid, the duration of air conduction is longer than when the usual Rinne test is done; in fact, it lasts seventy to eighty seconds. This is, of course, due to the fact that when the stem of the fork is pressed against the bone the excursions of the instrument are interfered with and the duration of vibration thereby shortened.

In another article he calls attention to the fact that when the Rinne test is carried out in the usual manner, and the fork no longer heard with the prongs held near the meatus, if the stem be then inserted into the auditory canal the fork is again heard via air, and usually for a period of about twelve seconds. This method I have used in only a few cases, and obtained similar results.

Since pressure of the stem against the meatal walls tends to diminish the duration of the fork's vibration, even this test does not show the full extent to which a fork may sound. Bezold claims that "we could really determine the length of hearing by air conduction only, if we were able to approach the drum membrane as closely with the prongs as we do with the stem of the fork."

Thus, while these facts are not new, it has seemed to me not wholly useless to consider them, first of all, because, as above stated, they seem to have escaped the attention of many, and, secondly, because it is often beneficial to test out the statements of others, with the idea of substantiating or refuting them.

Some have objected to the Rinne test as such, owing to the fact that by bone we determine the length of vibration of the stem, and via air that of the prongs, hence comparing the action of different structures. The answer to this is that the fork vibrates as a whole, and thus the stem and prongs sound equally long.

Then again, there is the objection raised to the method of testing by air alone, after having done the Rinne in the usual manner, on the ground that the fork would not each time be excited to the same extent. This error is obviated, it seems to me, by the technic employed. For quite a number of years I have in all tests (except where the very low forks are used) caused the fork to vibrate by a uniform method. In the thesis* read before this society some years ago, on "The Analysis of the Weber Test," opportunity was seized to emphasize the importance, at least from a scientific standpoint, of care and uniformity in doing the various tests.

To insure within reasonable limits equal application of force in exciting the a^1 fork, I always hold it at right angles to the body and allow a small pleximeter (armed with rubber pad) to fall of its own weight from a perpendicular position, striking the flat surface of the prong. By not using any force except that produced by gravity upon the head of the hammer, the personal element of variation in the impulse imparted to the fork is, I believe, almost entirely eliminated. If, indeed, there is a slight difference of a few seconds in the fork's vibration, this can have no material influence on the results. By this method, in testing the same individuals several times, as a matter of control, there is in intelligent persons rarely noted a difference of more than two to five seconds.

We can realize that many will contend that since the Rinne, as done in the usual way, gives us as good a ratio of the air to bone conduction as is needed for diagnosis, there is no necessity of further complicating the test by determining the air conduction alone after doing the regular test. However true this may be, it is equally certain that we do not in this manner really find out the actual or approximately actual duration of air conduction. But even if unnecessary for the ordinary practice of otology, is it not worth while to perform a procedure which at most requires one or two minutes extra in any case, and which may in time throw more light on the auditory function? At any rate, nothing can possibly be lost by doing this and recording the results.

These statements, of course, refer only to the positive Rinne, for in the negative one the full duration of air conduction has been found before the fork is placed on the mastoid.

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*Read before the Chicago Laryngological Society, February 21, 1911.

In the attempt to see whether this difference between the air conduction, as shown after the fork, is no longer heard on the mastoid, and that noted by air conduction alone, twenty-five examination records of pathologic cases and twenty-five with normal ears were tabulated.

An analysis of these data shows that in the one hundred ears thus examined the excess of hearing by air alone over that shown in the regular Rinne averaged thirteen seconds. In the pathologic cases (fifty ears) the average was 10.7 seconds; in the normal cases (fifty ears) it was 15.2 seconds. The least difference was five seconds in a few instances, and the greatest was thirty seconds (Case No. 40).

The percentage of increased air conduction, when all cases were considered (one hundred ears), was twenty-eight per cent. In the pathologic cases the average was twenty-seven per cent, some being as low as eight per cent, and some as high as sixty-six per cent (Case No. 9).

In the normal cases the average found was twenty-nine per cent, some cases showing as low as fifteen per cent, and one as high as sixty-six per cent (Case No. 40).

It is interesting to note that there is thus very little difference between the reactions shown in the pathologic cases of this series at least, as compared with the normal ears. It is true that of the socalled pathologic cases the majority were tubal catarrh, or slight nerve degeneration. Perhaps the study of a series of marked nerve lesions would show different results.

It is rather striking that in many comparatively young individuals with negative ear findings the fork is often heard only a short time via mastoid—e. g., twelve to fifteen seconds; whereas, the majority heard via bone from eighteen to thirty seconds. This phase of the subject will be discussed more fully some other time. Where there is no nerve disease, its explanation may rest on various grounds, such as a difference in thickness of the bone, variation in pressure with which the fork is held against the mastoid, some peculiarity of the patient, etc.

It may seem strange to note in the list shown in the table of pathologic cases that so many of the ears designated as having a tubal catarrh show so well marked a positive Rinne.

While the findings of all the tuning fork tests could not be put down in the space at command, let me say that in those cases the high pitched forks were well heard, showing that no involvement, at least to any appreciable degree, was present in the nerve.

To summarize, it may be said that the duration of air conduction is considerably longer than is ordinarily noted in the usual Rinne test. While this excess is not materially different as between the normal and pathologic cases shown in our series, the examination of a large number of cases, particularly those with marked nerve degeneration, may possibly show a different ratio between the air conduction, as shown in the Rinne, and that determined without first placing the fork on the mastoid process.

In conclusion, let us suggest that the Rinne test be in every case carried out in the usual manner; then, if the reaction is positive, insert the stem of the fork in the meatus and note how much longer the sound is appreciated. Following this, excite the fork with the same intensity as the first time, hold it before the meatus only, and note the results by air conduction alone.

As before conceded, this consideration may prove to be more academic than practical in value, but why not apply a routine which requires but a trifling amount of extra time, and may in the end bring forth enlightening data?

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PATHOLOGIC CASES.

No.	Name	Age	Condition of ears	a^1 fork on mastoid		a^1 fork via air after no longer heard on mastoid		a^1 fork via air only		Increased duration of hearing via air, noted in seconds	
				right	left	right	left	right	left	right	left
1	T. S.....	40	Tubal catarrh...............	20	30	65	60	70	70	5	10
2	E. J.....	33	Tubal catarrh...............	18	18	40	40	55	60	15	20
3	H. G...	56	Tubal catarrh and nerve degeneration	15	15	50	55	60	60	10	5
4	A. D. W.	33	Tubal catarrh and nerve degeneration	40	40	40	45	45	50	5	5
5	F. A. B.	65	Tubal catarrh and nerve degeneration	22	18	50	40	60	50	10	10
6	L. B....	23	Luetic nerve degeneration...	10	15	15	30	20	45	5	15
7	L. B....	60	Tubal catarrh and nerve degeneration	17	12	40	20	40	25	5	5
8	L. A....	29	Tubal catarrh...............	15	15	35	40	50	45	15	5

9	L. B....	19	Nerve degeneration.........	15	10	50	30	65	50	15	20
10	E. L. B.	19	Tubal catarrh...............	15	20	55	55	60	65	5	10
11	H. C....	38	Slight nerve degeneration....	15	10	45	40	55	55	10	15
12	A. E....	35	Slight nerve degeneration....	20	20	45	45	60	65	15	20
13	D. B. F..	69	Slight nerve degeneration and tubal catarrh	12	20	50	50	55	55	5	5
14	L. F....	16	Slight nerve degeneration and tubal catarrh	16	20	42	38	55	45	13	7
15	M. F....	21	Tubal catarrh...............	15	12	40	35	50	40	10	5
16	A. G....	42	Tubal catarrh...............	18	15	45	40	50	45	5	5
17	L. L....	21	Functional nerve degeneration	22	22	35	35	45	40	10	5
18	F. L....	47	Nerve degeneration..........	11	15	45	60	60	70	15	10
19	B. P....	22	Tubal catarrh...............	25	25	45	25	60	40	15	15
20	B. D. P.	6	Tubal catarrh...............	25	25	60	45	65	50	5	5
21	C. A. S.	52	Slight tubal catarrh.........	15	15	40	40	60	65	20	25
22	G. T. T.	25	Slight tubal catarrh.........	22	17	55	53	65	70	10	17
23	S. N....	54	Nerve degeneration.........	12	12	45	40	50	50	5	10
24	S. Y....	38	Tubal catarrh...............	22	20	40	30	60	40	20	10
25	E. F....	19	Otitis media chronica.......	15	20	25	45	35	55	10	10

NORMAL CASES.

No.	Name	Age	Condition of ears	a^1 fork on mastoid		a^1 fork via air after no longer heard on mastoid		a^1 fork via air only		Increased duration of hearing via air, noted in seconds	
				right	left	right	left	right	left	right	left
26	S. A....	19	Cerumen	20	20	45	40	60	50	15	10
27	A. B....	22	Cerumen	30	30	40	40	50	50	10	10
28	J. H....	50	Negative	20	18	62	50	75	65	13	15
29	C. G....	36	Negative	12	12	40	45	60	60	20	15
30	R. E....	26	Negative	15	15	45	50	55	60	10	10
31	E. S....	36	Negative	12	12	45	45	65	65	20	20
32	S. W...	30	Negative	18	24	40	50	60	60	20	10
33	S. H....	20	Negative	15	22	45	48	60	60	15	12
34	L. K...	37	Negative	12	15	45	45	60	60	15	15
35	W. F. L.	22	Negative	22	22	50	50	70	65	20	15
36	C. H. M.	26	Negative	20	28	55	50	65	60	10	10
37	S. T....	21	Negative	18	22	50	55	60	65	10	10
38	G. Y...	25	Negative	25	25	50	50	65	65	15	15
39	B. H. H.	23	Negative	18	18	40	55	60	65	20	10
40	J. V....	25	Negative	30	25	48	45	75	75	27	30
41	A. R. K.	25	Negative	20	20	60	60	75	80	15	20
42	R. W. C.	25	Negative	18	18	60	62	80	80	20	20
43	E. C. R.	23	Negative	25	25	60	60	80	80	20	20
44	L. A. S..	25	Negative	28	28	60	55	65	65	15	10
45	C. D. S.	24	Negative	25	25	50	50	60	60	10	10
46	G. G....	26	Negative	20	20	40	45	65	65	25	20
47	E. C....	25	Negative	20	20	55	55	70	70	15	15
48	S. R....	48	Negative	30	30	70	70	85	85	15	15
49	H. V...	25	Negative	25	25	60	60	75	75	15	15
50	H. S....	31	Negative	30	30	60	65	70	75	10	10

XXIV.

CHLOROFORM AS A GENERAL ANESTHETIC IN NASOPHARYNGEAL SURGERY.

By W. H. Nardin, M. D.,

Anderson, S. C.

Chloroform as a general anesthetic has for many years been held in great disfavor by physicians and surgeons in America, the belief that it is a dangerous drug in that capacity having so widely prevailed as to cause its use to be generally abandoned in the practice of surgery. Some states have taken so grave a view of its risks as to pass restrictive laws regarding chloroform. I must, in consequence of these facts, beg for these modest observations of mine an unprejudiced reading upon the part of those into whose hands they may fall.

We, as doctors, are often reminded that, despite the remarkable revelations of the past fifty years, medicine is not an exact science; hence the spirit of investigation often prompts us to go back and try again remedies, both medical and surgical, that have at some former time been rejected; and not infrequently the retesting gives us a better acquaintance with, and a clearer understanding of, these remedies in their uses as well as limitations.

Remembering that chloroform, although it has been discarded by most American surgeons, as a general anesthetic, is still conceded by all to be the best anesthetic in obstetric practice, despite the fact that a pregnant woman, particularly in the later months of pregnancy, is a poor surgical risk, I decided to give it a trial in my work, about six years ago. I have used it continuously since in all cases in that class of work where a general anesthetic was required, and out of approximately a thousand cases have not had one fatality, though some have come very near the borderline. But what operator does not encounter more or less trouble with an anesthetic when he cannot have at all times an expert anesthetist, but is forced to use country practitioners with varied and, for the most part, poor training in the giving of anesthetics?

I have watched carefully the results of my work, and am convinced that chloroform, when properly given, is as safe as any other anesthetic for this class of work.

Furthermore, it possesses some advantages to which I would like to call attention. A very small amount of the drug will produce anesthesia, about two drams being sufficient for the operation for removing tonsils and adenoids, this being the maximum amount for the average case. The congestion is less about the head and neck, consequently less hemorrhage; there is less mucus in the throat, which is a great advantage in this class of work, since a clear field helps so much, both in the time consumed during the operation and in the ability to avoid injuring the surrounding tissues.

The shock is less, as indicated by the blood pressure, and its rapidity in recovering its standard.

The lowering of the blood pressure is less and is more fully gotten to normal, which is a distinct advantage in postoperative complications, as the normal resistance is not so long interfered with.

The stage of excitement does not last as long, nor is it so well marked as with other anesthetics; as a rule the patient goes to sleep as quietly as in his own bed.

It is much pleasanter to take, as there is less of the feeling of suffocation. This is very desirable with children, for they are as a rule not alarmed by it at the very beginning, and we all like, if possible, to avoid this condition.

It takes less time to anesthetize the patient, and, because of this fact, one accustomed to using ether will have to be especially watchful. In my practice the patient is usually well under in fifteen minutes, or sometimes in less time; that is, the corneal and pharyngeal reflexes have completely disappeared.

The duration of the anesthesia is not so extended, and the patient comes out more quickly. This is of especial advantage when the operation is performed in a private office, for, in a short time, approximately half an hour, the patient can be carried home. And I find that being carried into the fresh air, they more quickly recover; even if it is cold weather, if they are well wrapped, there is no danger of bronchial complications, as when some other anesthetics are used. Patients sometimes go as far as fifty miles through the open country, the only inconvenience being the nausea and vomiting from the amount of blood swallowed during the operation. However, there is less nausea than usually follows any other anesthetic; nor does the nausea last as long.

There is less liability to postoperative complications, viz., bronchitis, nephritis, or pneumonia; for when the patient fully recovers from the immediate effects, one can feel that they are entirely safe.

Chloroform, since it can and must be given well diluted with air, requires no special or complicated apparatus, and can, therefore, be used in an emergency at any time or place by anybody; but to be of advantage to both patient and operator, it should be properly given in conformity to the following method:

First. Have an anesthetist who is in the habit of administering chloroform, one who will be directed during the course of the anesthesia, for most of the accidents from chloroform come from the one who goes to sleep on the job—and a careless anesthetist is more dangerous than the drug.

Second. Use only chloroform that you know to be chemically pure, and it must not be old.

Third. Always give it on an open mask, so that it will be well mixed with air.

Fourth. Give it drop by drop; never crowd it in the least; and the operator should never hurry the anesthetist, for the result may be disastrous to both the patient and the surgeon.

Fifth. The anesthetic should, when possible, be given in a room detached from the operating room, where the rattle of instruments an bustle preparatory to the operation will more than likely excite the patient, which we wish to avoid.

If these simple rules are carried out, it will be found that the drug is not so danger-fraught as we often suppose, but has many advantages of its own.

When the drug has not been taken well by the patients in my series, the following symptoms are noted: When I thought the patient should be fully anesthetized, there is some struggling, the pupils dilate, there is a sudden stoppage of respiration, slowing of the pulse, cyanosis, abolition of all reflexes.

The following treatment has been used effectually: Lower the head; open all windows, use artificial respiration, give hypodermic of strychnin, and adrenalin chlorid solution, and in some cases use oxygen gas.

SUMMARY.

For the removal of tonsils and adenoids, when a general anesthetic is necessary, chloroform is as safe as any other, and has much in its favor; that it must be given slowly and well diluted with air; that the danger comes from crowding it, by the operator or anesthetist being in too great a hurry; that the respiration stops before the heart, and that this stoppage comes on suddenly.

I write this with the hope that some observers more competent than I will test out my simple plan here suggested, and see whether or not we are neglecting a most valuable aid for lack of courage on our part.

"He who fights and runs away
Will live to fight some other day."

XXV.

A NEW MASTOID FLAP.

By Adair Dighton, F. R. C. S.,

Liverpool.

Having for years past struggled, with varying success, with the multitudinous flaps, described in the textbooks and journals, for the filling in of the postoperative mastoid cavity, it seems to me to be worth while to describe yet another, as it has the merit of simplicity, and in addition preserves the periosteum which, except in a few cases, has been entirely neglected.

The skin incision that I use is the classic one, extending from a point half an inch posterior to the temporal artery above and curving downwards about a quarter of an inch behind the attachment of the auricle, to finish at the center of the tip of the mastoid process. This incision goes through skin and subcutaneous tissue only, and during its performance the auricle is pulled strongly forward and dissected, almost peeled, from the underlying pericranium until the cartilaginous meatus is reached. The exposed pericranium is then incised by a cut extending from a point just behind the suprameatal spine upwards and backwards to a point just anterior to the line of the original skin incision. A second incision extends from a point just behind the lower angle of the cartilaginous meatus downwards and backwards, diverging from the first, to a point just within the line of the original skin incision. A third incision joins the posterior ends of these two, running in the same line as, but just anterior to, the original skin incision.

The pericranial flap, so formed, is raised from the bone and pushed forwards and held out of the way by the anterior plate blade of my selfretaining mastoid retractor, which is inserted between the pericranium and posterior osseous wall of the canal. The mastoid operation, whether Heath or radical, is then performed, and at its completion the retractor is removed and the pericranial flap clipped and held with a pair of catch forceps. Next, a flap is made in the posterior wall of the cartilaginous meatus, after the method of Körner. The first incision is made along the junction of the posterior and superior walls of the canal and above the attached pericranium, whilst the second incision is made along the junction of the posterior and inferior walls of the canal and below the attached pericranium. The next step is to raise the pericranium along the posterior margin of the wound and, also, to separate it as far as possible from the overlying skin and subcutaneous tissue. This done, a catgut stitch is passed through the upper angle of the pericranial flap, and the two threads passed on a single needle through the upper angle of the raised posterior pericranium from within outwards. The same process is repeated with the lower angle of the flap to the lower angle of the raised posterior pericranium. The stitches are then pulled upon and the pericranial flap drawn under cover of the raised posterior pericranium and the stitches are then tied, the upper two threads to the lower two. In place of this catgut stitch a silkworm gut stitch may be passed through the pericranial flap from without inwards, and then from within outwards, and the two threads passed on a single needle under the raised pericranium and out through skin about half an inch behind the posterior edge, and tied tightly over a tube or roll of gauze.

Now, the Körner flap has been so cut, above and below the attachments of the pericranial flap to the cartilage of the meatus, that when the stitches, whether catgut or silkworm gut, are tied, the attached pericranium pulls on the tongue of the cartilaginous flap, draws it back into the postoperative cavity and holds it in position in identically the same way as it would be drawn back and held if the flap itself were stitched.

It has a distinct advantage in that it forms a pericranial covering to the cavity, and being in close proximity to healthy periosteum becomes united to it, and so hastens healing. The posterior skin incision is, of course, stitched in the usual way.

XXVI.

PAPER CLIP IN BRONCHUS SEVENTEEN YEARS, REMOVED BY SUPERIOR BRONCHOSCOPY.

By Edwin Beer, M. D.,

New York City.

During the past seventeen years numerous cases of foreign bodies removed from the bronchus by either the superior (laryngeal) or inferior (tracheal) route have been recorded. The following case is placed on record because of the long interval* between aspiration and removal, and to emphasize the fact that even after remaining so long in situ its removal was not difficult.

M. K., aged twenty-six years; admitted July 1, 1915; discharged July 19, 1915. History of present illness began seventeen years ago, when patient was playing with a paper clip which he had put into his mouth, and it suddenly became lodged far back in his mouth near the base of his tongue. His father, with the use of an ordinary teaspoon, made an attempt to remove the clip, but unfortunately pushed it still farther down the patient's throat. The patient said that he felt much better and did not notice any particular distress, and his parents believed that he had swallowed the clip, whereupon he was given castor oil. However, the next day, while the patient was playing in the street, he was suddenly seized with a paroxysm of coughing and raised a large quantity of mucus mixed with blood, also having great difficulty in breathing freely.

He was taken at once to a hospital, where he was placed under observation for a period of one week. After being put to bed his symptoms disappeared, and he was finally discharged without any attempt being made to recover the paper clip, of which he says he told the doctor.

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*Chevalier Jackson, Annals of Otology, Rhinology and Laryngology, June, 1913, reports a remarkable case in which he removed a collar button twenty-six years after aspiration.

After being home for a few days he was taken suddenly very ill, and his physician said he had pneumonia. He was sick at this time for about six weeks, having had what the doctor called a relapse, from which he finally made an uneventful recovery. He then enjoyed fairly good health for about two years, when he noticed a dull aching pain in the right side of his chest, and complained of a hacking cough with expectoration of large quantities of mucopurulent sputum, marked shortness of breath, and a sense of oppression and suffocation. Thinking that he was beginning to have some lung disease, he presented himself to his physician for examination. He was first told he had tuberculosis, and must come back for a second examination after a certain period, which he did, and was then told he had bronchial asthma. He received treatment for this last mentioned condition for a period of about twelve years in several institutions and from many physicians, but without relief. During this time he coughed more, sputum became very offensive and foul smelling, his pain and sense of suffocation increased, and he became exhausted on the slightest exertion, finally having to give up his work entirely. His weight, however, did not decrease with increase of his symptoms. His appetite was always good. Not being able to work, he gave up all hope and came to Bellevue Hospital, believing that he had tuberculosis, and applied for admission. Upon being questioned, he told of the paper clip he had had in his mouth seventeen years ago, and of the treatment he had received since that time. He was admitted to the hospital for X-ray examination on July 1, 1915.

Complaints on admission were:

1. Dull aching pain in right side of chest.
2. Paroxysms of prolonged coughing.
3. Expectoration of increasing quantities of foul, offensive, grayish sputum.
4. Shortness of breath, sense of oppression and suffocation.
5. Loss of strength.
6. Insisting that his trouble was due to the paper clip, which he said was in his lung.

X-ray examination showed a very distinct shadow of a flat-headed paper clip in the right main bronchus, with head down. It also showed marked thickening of the adjacent pulmonary tissue.

July 15, 1915. Superior bronchoscopy and removal of clip under combined ether and cocain anesthesia from right main bronchus. Cocain and adrenalin were applied in right bronchus to control reflexes, and ether insufflation (intratracheal) through metal catheter in the bronchoscope kept the patient thoroughly anesthetized. The clip was imbedded in the granulation tissue which almost filled the bronchial lumen, and there was copious discharge during the manipulations. As soon as one leg of the clip was seen it was firmly grasped with forceps and the clip withdrawn with bronchoscopic tube (Jackson-Rochester). In withdrawing the clip the two legs became spread apart, one entering the tube and the other trailing after.

July 17, 1915. There was very little febrile reaction. At first expectoration ceased almost entirely, and then it became more copious and then gradually diminished in amount.

July 19, 1915. Discharged feeling very well.

SOCIETY PROCEEDINGS.

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLOGY.

Meeting of December 16, 1915.

Orientation and Equilibration: A Study of the Sense of Position and Movement: Its Dependence on the Vestibular Apparatus and Its Importance in the Whole Field of Medicine.

DR. ARTHUR B. DUEL: After referring to the vast amount of literature that had accumulated with reference to our knowledge of position in space and adjustment to it, Dr. Duel spoke briefly of the work of Flourens, in 1828, who first demonstrated that the semicircular canals were definitely connected with the movements of the head; of Schrader's experiments on frogs; and of Professor Lee's experiments on sharks, which hardly left any doubt as to the participation of the semicircular canals and the labyrinth in the equilibration process. These and other physiologists had furnished proof that the vestibular apparatus is a special sense organ, solely concerned with orientation and equilibration; that its physiologic connection with the auditory apparatus is practically nothing compared with its intimate connection with other special senses, such as sight, the kinesthetic sense (muscle-joint), and the sense of touch. Hence, the main object of this paper was to bring out this fact, which was not generally appreciated, namely, that the eighth nerve, the auditory, should be described in anatomy and physiology as the nerve of hearing only, with its end organ, the cochlea; the socalled vestibular branch should be given a place by itself, and should be described as the nerve of orientation only, with its end organ, the vestibule and the semicircular canals. So far as the anatomic evidence in the matter was concerned, it might be observed that the distribution of the vestibular and auditory fibers was to entirely different centers. One had but to contemplate for a few moments the various precise poises of the body in space, together with the adjustments of the limbs and other parts to the accurate localization required in daily existence, to be deeply impressed with the nicety of the mechanism which controlled them. The development of these adjustments to meet the demands of increasing activities was very rapid. A normal child of seven or eight years had unconsciously acquired a sense of orientation, by an adjustment of muscles and joints, which in magnitude far exceeded that acquired in later years by practice and training for the most difficult pursuits. This particular function of orientation was more important than any other in the struggle for existence. Its evolution might be traced from the stiffened tentacle of the jellyfish, through the calcareous particles of the higher forms to the more elaborate vestibular apparatus of the vertebrates, in which it became more important in all the acts of the individual. As the higher order of beings was reached, the sense of position was safeguarded and augmented by intimate association with other sensations, sight, the kinesthetic sense, hearing, touch, smell, etc. In the perfectly normal individual the conception of position was the result of the harmonious assembling of all these afferent impulses. Loss or imperfect action of any of them produced a disharmony which, if not corrected, resulted in a misconception varying with the degree of the upset. There might be simply a slight uneasiness or uncertainty at the mere thought of some daring act of equilibration, or some previous experience like looking from a dizzy height, a rough sea voyage, a ride on a merry-go-round, or other psychic illusory idea. Again, there might be a varying degree of vertigo resulting from the actual experiences, the thought of which might cause the psychic vertigo, and on to the more violent manifestations of vertigo accompanied by nausea, vomiting, loss of equilibrium, and ataxia. Through all this one should not lose sight of the fact that the vestibular apparatus was the most essential factor in the sense of orientation, the only source of such afferent impulses as had to do with that sense alone. The phylla deprived of its stiffened tentacle, the higher forms deprived of their otoliths, completely lost their sense of position and could not maintain existence. In the human, in whom the struggle for existence was the fiercest, it was quite natural that the compensatory factors for the loss of this sense of position furnished by the vestibular apparatus should be more complex. A man deprived of one vestibular apparatus was temporarily upset in his sense of position; as a result he had violent vertigo, which his other senses made frantic efforts to correct. The violent effort to correct a change of position which was not actual, resulted in loss of equilibrium. These efforts to compensate for the loss of a vestibular apparatus succeeded with varying rapidity. The more violent manifestations subsided in a few days, and in a few weeks the ordinary acts were performed with apparent ease. However, in such a case the orientation could not be said to have become perfect again, but the individual had simply adjusted himself to the loss. Perfect orientation required angulation; impressions must come from the two different vestibular apparatuses at the same time, and the individual had learned the trick of moving his head or eye to get a point of view from two slightly different angles at practically the same instant. The deafmute born without vestibular apparatus, did many acts of equilibration with perfect ease and might miss many of the upsets of life which those with perfect orientation might have. He might never experience the throes of mal de mer. Nevertheless, the loss of both static labyrinths was a great catastrophe. While the first symptoms might not be violent, the task of learning to orient without the aid of organs specially designed for it was much like a blind person learning to read from the sense of touch, or the deaf one interpreting the movements of the lips from the sense of sight, and often the results were quite as imperfect.

To appreciate this one had only to try any of the more difficult acts of equilibration on such a patient. For instance he might be asked to try to stand on one leg, with the eyes closed, to walk forward or backward with the eyes closed, or an act of walking or turning in the dark. The normal individual might have the illusion when sitting in a train at a station that the train was moving, when, in reality, it was a train by his side that was coming in or going out of the station. This optical illusion was quickly corrected by the normal individual by looking at some upright portion of his own car which was not moving, or by closing his eyes, when the optical illusion was corrected by the static labyrinth. If a congenital deafmute were to have this illusion, it would have to be corrected by the sense of sight. If he closed his eyes the illusion would remain, particularly if there were no vibration which would upset his tactile sense. The essayist stated that in having introduced this subject in a somewhat desultory manner he hoped he had succeeded in showing how orientation might be upset by misinformation from many sources; how impulses from the static labyrinth were so powerful to correct the misinformation, and, on the other hand, how misinformation from the static labyrinth itself was corrected with comparatively great difficulty by other afferent impulses. He would leave it to those who followed to point out the methods of fixing the cause of disequilibrium on peripheral or central organs, on functional or organic disturbance, on lesions of the different afferent sources of information, on the cerebellum, or the cerebrum, which, after all, must be the source of our consciousness of position as well as that from which we voluntarily changed it.

DISCUSSION.

Dr. F. H. Pike: I must confess to a certain hesitancy in speaking to you on anything apparently so well known as the otic labyrinth. The subject is so well known that the man in the street immediately associates the otic labyrinth and the cerebellum with the maintenance of equilibrium. And so firmly is the belief that the cerebellum is the great central mechanism for the maintenance of equilibrium fixed in the mind, that there is a strong tendency in some quarters to assert that the disturbances following lesions of the cerebellum and lesions of the labyrinth are essentially similar. But if we are to distinguish in clinical diagnosis between lesions of the labyrinth and lesions of the cerebellum, it appears to me that it is far more important to point out certain constant differences in the effects rather than to dwelling so strongly upon the resemblances.

Our knowledge of the effects of lesions of the labyrinth and of the cerebellum is derived partly from clinical observation and partly from experiments. We may point out that the difficulty of limiting the effects of a pathologic lesion to the area primarily involved is rather great. We may have, first, the effects of pressure resulting from an intracranial growth and also the increased secretion of cerebrospinal fluid which usually accompanies an intracranial tumor, and which will in its turn give rise to abnormally high pressures. This fact was announced from this platform about a year ago by Dr. Charles H. Frazier. In the case of intracranial or mastoid abscesses we have the spread of toxins and other effects upon remote parts of the nervous system. Secondly, there is the difficulty of getting human material for postmortem examination at the time when such examination would be most desirable for the pathologist.

The necessity for the experimental method of attack lies in the necessity of overcoming, in part at least, these difficulties. In the experimental method we may, if we so desire, avoid the effects of increased intracranial pressure and bacterial toxins upon other parts of the nervous system. Further, we may control the extent, both of the primary and of the associated experimental lesions, within certain limits. One must recognize, of course, that when a nerve fiber is separated from its cell of origin a degeneration will proceed to the furthest limits of the fiber, and to this degree the extent of the experimental lesion may be beyond our control. And finally, the experimenter may control the length of life of his animals, and get his material for postmortem examination at the time which he considers most opportune or advantageous. As an experimentalist I would, therefore, urge upon you the need for greater collaboration between clinical and laboratory workers. It is not my idea to supplant clinical observation, but to supplement it experimentally.

The lantern slides which were shown illustrated the effects of lesion of the labyrinth and of the cerebellum upon a considerable number of animals. These figures, for the most part, have been published in the Philosophical Transactions of the Royal Society of London, 1912, Series B, Vol. 203, pp. 127-160, and in the supplement to the Transactions of the American Otological Society, 1914, Vol. xiii, part II, by Prof. J. Gordon Wilson and myself. In all animals so far studied the effects of experimental lesions of the labyrinth result in a torsion of the head to the injured side. Immediately after the operation the animal may turn or roll over and over to the injured side. When both labyrinths are removed at once the animal—e. g., a dog—may refuse to stand unassisted, but lie with the limbs and head as closely applied to the floor as it can get them. The animal learns to stand after a few days, and to walk about without falling. But even months afterward it will be unable to jump from even a moderate height to the floor without falling in a heap. This observation is originally due to Schiff, and we have repeated it many times. There are always certain deficiencies of this kind which may be discovered by careful examination of animals from which the otic labyrinths have been removed.

When one looks at the central nervous system from the point of view of its evolution, it is seen that in the lower forms of vertebrates the cerebral hemispheres form a very inconsiderable portion of the whole nervous system. But as successively higher animals are taken, we find that the anterior end of the nervous axis—that is, the cerebrum—shows a greater and greater degree of development. Experimental findings agree very closely with the anatomic development, and all the facts so far gathered point to the increased importance of the cerebrum in the daily life of the animal. And on the basis, both of the anatomic development and of the considerable number of experimental facts, it is our belief that one must look more and more to the cerebrum as the great central mechanism for orientation in space, rather than to the cerebellum.

DR. WHITNEY said that it would have been difficult for the writer of the evening's paper to have selected a subject for presentation before this audience which would more thoroughly have tested his powers of analysis, his originality of observation, and his fairness of judgment. Nor could he, in his opinion, have offered a contribution more timely in its application to our present medical needs, or of greater practical importance to the entire medical profession.

Why is the subject chosen so difficult of practical treatment? Because it involves an explanation of complex conceptions and confusing manifestations which are of almost boundless variety and extent, and because it requires the reconciliation of numerous conflicting theories, many of which play an important rôle in any explanation which may be offered as a solution of the perplexing problems of equilibrium and orientation.

No elaborate argument is required to establish the indisputable value in the scheme of our daily life and activities of the function of equilibrium; it must, indeed, be regarded as second only in importance to those vital processes, respiration and circulation, the interruption of which will speedily terminate the life of the individual.

How primarily essential to the existence of the animal organism is a knowledge of its position in space becomes the more readily appreciated if we permit ourselves a short excursion into the field of biology, the teachings of which science have clearly established that all forms of multicellar life present purposeful or somatic reactions which result in controlling their attitude toward or away from any source of irritation, in consequence of the possession of which faculty these organisms are able to select the most favorable position in which to place themselves.

In this appreciation of its relation to space, as indicated by recognition of restraint or freedom of motion, we may consider that we find our earliest suggestion of the function of orientation.

That an organism existing under primordial conditions should find some method, however simple, of determining its position in space essential to its survival, naturally suggests to the scientific mind a proposition which may be formulated in the following law: namely, in the progress of evolution through the succeeding higher grades of differentiation of structure, there must inevitably be found a development of this faculty (the sense of position) proportioned in its intricacy to the complex conditions governing the environment of this higher organism.

Such are in fact the logical steps whose orderly succession following through the lower organisms, thence upward to the human being with its highly specialized central nervous system, has resulted in our present conception of our position in space or orientation.

With increased complexity of structure and differentiation of the body form into head, trunk and extremities, there is developed a more accurate sense of balance, whether at rest or in motion, associated with a well defined sense of direction.

Thus, little by little, through an infinite series of gradations keeping pace with the physical needs requisite for the preservation of the animal, the sense of adjustment to surroundings is formulated, and under the stimulus of the will and intelligence at length fully established.

Intricate as are the physical sensations and nervous impulses, the correlation of which are required for the maintenance of equilibrium among the higher animals, they still are wanting in many of the complex conceptions which constitute essential factors in the function of orientation in man.

So remarkable are the functions attributed to the vestibular apparatus in its relation to equilibrium and orientation, as to suggest that this organ may properly be regarded as the guardian of our sixth special sense.

That the function of equilibrium is a congenital possession of the human being, there seems every reason to doubt; for it is common observation that the infant, although strong enough to stand firmly on its feet, and to jump vigorously when supported, cannot maintain its equilibrium unassisted until it has gradually mastered the difficulties of equilibrium.

In orienting himself a man readily finds his relation to an object that he can see by employing the aid of his visual powers, to an object that he can feel by the aid of his tactile sense, and to the direction and source of sound by the function of his auditory mechanism; numerous additional afferent sensory impressions, however, contribute to the establishment of effective orientation. Of the above mentioned factors, each bears an important relation to its fellow in the function of equilibration, and the derangement of any one element of the group is sufficient to disorganize the correlation of all; it is, however, in disorders of the vestibular apparatus that we encounter the most severe and sudden disturbances in equilibrium.

The infinite complexity of the afferent impulses, whose harmonious correlation is essential to orientation, have been clearly indicated by the writer of the evening's paper, and I shall not weary you with attempts at recapitulation, merely confining my further remarks to a brief comment upon those manifestations of disharmony with which, as clinicians and otologists, we are interested, and which ensue upon a disturbance of those components of equilibrium over which the vestibular apparatus presides.

The history of systematic and scientific investigation of the physiology of the inner ear, and especially of the complex function of equilibrium, dates back far over a century.

The questions involved are so intimately related to other chapters of physiology, notably of the visual perception, the sense of space, direction and position, and are so often suggested by the practical experience of the clinic and operating room, that observers in the most widely distributed fields have contributed to our present knowledge.

It is, however, only within the last decade that the widely diversified and oftentimes fantastic views entertained regarding the complex functions of the various organs concerned in establishment of equilibrium have received proper interpretation, and even at the present moment there remains an embarrassing array of incompatible theories and hypotheses, each with its enthusiastic advocates, which are absolutely irreconcilable upon the basis of established knowledge.

Whether we can accept the views that the stimulation on impulses in the static labyrinth are aroused by gravitational pressure, or are the result of the movement of columns of fluid (endolymph), is not essential clinically to our interpretation of the manifestations which ensue in consequence of irritation of the vestibular apparatus, however interesting such studies may prove to the student of physiology.

The writer of the paper has considered in its broadest physical application the significance of equilibrium and orientation as related to the vestibular apparatus, and some of his suggestions will, I anticipate, provide food for mature reflection for our neurologic colleagues.

Of such a nature is the perplexing problem presented by the deafmute, who with the destruction of both labyrinths develops a compensatory sense of equilibrium largely dependent upon visual impressions supplemented by the tactile and kinesthetic sense; his orientation, however, always remains defective. Such a person finds it impossible to maintain his sense of direction when swimming under water; moreover, owing to the bewilderment of his faculties from loss of orientation, a deafmute, although a capable swimmer on the surface, would be in danger of drowning from his own misdirected efforts, if he were suddenly plunged into deep water.

If one would appreciate in a slight degree the difficulties of orientation when undertaken without the aid of sight and hearing, let him close his eyes, hold his fingers in his ears, and attempt to walk straight ahead for any considerable distance.

He will begin his walk with assurance, lifting his feet for a few steps with his usual confidence, but before he has proceeded many paces he will begin to slide or push his feet along the floor, instead of lifting them, in order that he may preserve a better grasp on his position. His stride will shorten and his mode of progression gradually degenerates into a lumbering sort of shuffling of the feet, finally terminating in a loss of sense of position which brings him to a halt.

He experiences these difficulties, notwithstanding that he is assisted in his experiment by a normally functionating vestibular apparatus.

A person suffering with any structural vestibular changes would exhibit an exaggeration of these various manifestations, and would deviate toward the affected or weaker side, and with the application of von Stein's test, the disability would become progressively more apparent.

To the practitioner of medicine an enumeration of the complex array of motor and sensory phenomena, whose harmonious adjustment or correlation results in the function of orientation, is of interest chiefly as an exposition of a scientific demonstration; but he attaches far more importance to one practical suggestion which will enable him to treat intelligently the manifestations which result from the disturbance of this adjustment, and to which we have applied the name of vertigo, than to the elucidation of any theory, be it never so ingenious.

His vertiginous patient is usually a practical and insistent person, who demands assurance that his distressing symptoms will soon be relieved, and the doctor must have at his command an understanding of the condition which will enable him to decide whether the case is one of functional derangement which will soon right itself, or whether he is dealing with structural changes in the vestibular apparatus, nerve or cerebellum, from which he may apprehend a permanent disability or even a fatal termination.

Can we, then, indicate any definite diagnostic procedure which will clearly establish the causes of disturbances of equilibrium or vertigo in any given instance.

From the clinical standpoint of the otologist, we are accustomed to encounter vertigo in connection with labyrinthine inflammations, meningitis, abscess or tumor of the cerebellum, and vestibular hemorrhage and hysteria.

In the first mentioned condition of infective labyrinthitis, except in the event of a metastatic inflammation (an exceedingly rare manifestation), the presence of suppuration of the ear would at once indicate the source of the disturbance, while the concomitant nystagmus and deafness would supply the needful corroborative evidence.

While in otitic meningitis the history of aural suppuration with rigidity of the neck and the presence of Kernig's or Babinski's signs, when supplemented by the information afforded by spinal puncture, will ordinarily establish the diagnosis.

With abscess of the cerebellum we are again guided by the presence of ear suppuration, but with cerebellar tumor there may be no evidence of ear disease. The vertigo associated with these lesions is usually attended by cerebellar ataxia, while the disturbances of balance, instead of diminishing in intensity with the duration of the disease, are disposed, in contradistinction to labyrinth inflammations, to increase in severity. Again, with abscess or neoplasm of the cerebellum, the direction in which the body falls is independent of the nystagmus if this is present, and does not change with altered position of the head; in addition to which evidence the "pointing-by" experiment should indicate a retrolabyrinthine affection.

Hemorrhage of the labyrinth is attended with complete loss of hearing and with vertigo of such violent character as to preclude all attempts at observing the reaction movements; however, the history of sudden deafness, with attendant vomiting, vertigo and characteristic vestibular nystagmus, will readily indicate the diagnosis.

With attacks of hysteric vertigo the middle ear on physical examination may show no pathologic changes, while nystagmus, if present, is not of the vestibular type, nor are the disturbances of equilibrium such as are caused by labyrinthine disease; the body may have a tendency to fall backward or forward or to either side—that is, it follows no definite rule—while in labyrinthitis the tendency is to fall always in the direction of the slow component of the nystagmus, whatever the position of the head. In hysteria, deafness may be simulated, but the static labyrinth will react to functional tests. (Braun-Friesner, p. 200.)

The study of the function of the inner ear and the logical systematizing of functional test is by no means complete, but enough has already been accomplished to show that we are progressing in the right direction.

No doubt, an elaboration and critical analysis of all rationally scientific methods will be of eventual service to the practitioner, and the final result of accurate and painstaking investigations in laboratory and clinic will bring theory and practice into much desired accord and correspondence.

The objections which have been raised to the errors inherent in this or that test, the exception to minor points of this or that theory, are details which are in nowise fundamental and cannot invalidate the principles involved, nor prevent their eventual application. In fact, the practical objections of competent clinicians and surgeons can serve only a beneficent purpose in correcting errors while they stimulate anew the search for truth. (Friedenberg, "Non-Acoustic Functions of the Labyrinth.")

Paper: The Role of the Eyes in Equilibration and Orientation.*

BY ARNOLD KNAPP, M. D.

Paper: The Importance of the Vestibular Mechanism in Neurology.†

BY J. RAMSAY HUNT, M. D.

DISCUSSION.

DR. FRISSELL: The rôle of the general practitioner in discussing the subject of vertigo, especially after listening to the brilliant expositions from the special points of aurist, physiologist, ophthalmologist, and neurologist, is a trifle difficult. Why is he here at all? The answer seems to be that, as ancient Rome gave her successful general a triumph with captives at his chariot wheels, so our First Consul has consented to accentuate the papers of the evening by insisting on the violent contrast of an internist discussing the specialist's pet field.

The general man is, however, deeply concerned, in that to his inexperienced hand comes the patient seeking relief from real symptoms, and here comes both his perplexity and his duty—to which branch of the dazzling array of specialists should the patient be referred? Is it a case of labyrinthine vertigo, and is the aurist to be called upon; or is muscular weakness of the eye the predominant factor? Is it a cerebellar tumor or a disease of the central nervous system which demands the special attention of the neurologist? Or, more difficult still, is it one of those threshold cases on the borderland of epilepsy, simulating labyrinthine disease, so admirably described by Gowers?

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*See page 453.

†See page 446.

Fortunately, the dissentions on the internal groups somewhat help the perplexed practitioner. Meniere's original description of auditory vertigo was of a symptom complex, of sudden vertigo associated with nausea, vomiting, and deafness on the affected side; in other words, a disease showing the same symptoms as our experimental animals in which the semicircular canals of one side have been extirpated, the reasons for which have been so admirably described by my predecessors.

But gradually it was found that all cases did not have either the classic symptoms of Meniere's disease, or the classic cause —destruction of the labyrinth—but varied in intensity from slight tinnitus with vertigo to the severe type originally described. So arose the classification into Meniere's disease and pseudo-Meniere's; the former having as its pathologic base the destruction of the labyrinth, while the latter was attributed to slighter forms of disease, such as otitis media, changes in pressure of the endolymph, cerumen in the external meatus, disturbances in the vestibular nerve in its course to the central nuclei, or even more remotely to tabetic changes and the functional explosive irritation of the higher centers in the epileptic aura and attack.

But if vertigo may be attributed to so many diverse sources in which the ear is affected directly or indirectly, what of those cases in which muscular incoordination in the eye produces diplopia and a false and uncorrected concept in the brain, the vertigo of multiple sclerosis—that psychic type caused by looking down from a height in one unaccustomed to mountaineering; the common incoordination of acute alcoholism, and that distressing form met with in "mal de mer"? Must the general practitioner be relegated to the undignified position of

"Mother Sill," and be altogether eliminated from the diagnosis and treatment of this common malady?

In its ultimate analysis, the causes of these symptoms are in many, if not in most, cases a distinctly medical affliction, as syphilis, arteriosclerosis, aortic insufficiency, the various types of anemias, diseases of the gastrointestinal tract, which are directly responsible, not only for the appearance of the symptoms, but in many cases for the pathologic base of the symptom complex, the latter forming only a localizing symptom of a constitutional disease.

One must conceive it to be the duty, then, of the general practitioner, in the presence of vertigo, by careful examination of his patient—including particularly blood, urine, blood pressure, and serologic examinations—to first determine the presence or absence of underlying constitutional disease; and, secondly, by careful examination of the eye, ear, and nervous system, to determine in which special field the immediate defect causing vertigo lies. If, as is probably the case, he is himself unable to apply the more complicated rotation and caloric tests in the ear, or make a thorough examination for muscular strain or defective vision in the eye, he should refer such a case to the better trained specialist for a more thorough examination.

But with his broader field of vision, he should be able to coordinate the examinations of his technically more skilled confréres, and will often be enabled to arrive at a more correct diagnosis in a difficult case.

In treatment, too, where a cure may not be obtained by a correction of muscle palsy, a regulation of pressure in the labyrinth or by operative procedure, his handling of the hypersensitive and anemic conditions may greatly improve, or in syphilis brilliantly cure, the underlying conditions.

Therefore, the internist's plea is that even in this field he may be allowed some slight recognition from his more skilled brethren, and may be able to retain his place in the care of so obscure and puzzling a condition.

Dr. Joseph Byrne said that a distinction should be made between equilibration and orientation. Equilibration is a means of orientation, but must not be considered as the equivalent of orientation. He was pleased to hear Dr. Pike say that we must look ultimately to the cerebrum for the mechanism of disturbances of orientation following injury of the labyrinth.

Orientation, in general, may be said to mean the perception of one's relation to his environment. An old definition of truth is that it is conformity between thought and thing. Orientation may be defined as conformity between the immediate testimony of our senses anent our relations in space, and our conception of what those relations are or ought to be. We have, then, in orientation two fundamental elements: (1) the immediate testimony of our senses, and (2) our conception, based on education and experience, of what our relations in space under any given set of circumstances ought to be. It is evident that the second element is mainly psychologic, and forms the background for the interpretation and evaluation of the first element—i. e., of present incoming sensory impulses from all sources.

Insofar as element number one is in accord with element number two, insofar are we orientated. Conflict to a certain degree between these elements causes disorientation—i. e., uncertainty of one's relations, with consequent sense of insecurity, fear, etc. The labyrinth is no more the organ of orientation than many other sensory mechanisms—e. g., kinesthetic, visuoocular, tactual, etc.—which inform us immediately of the actual relations of our bodies in space. But information from these sources has to be integrated and evalued against the background of experience before they enter into the mechanism of orientation. Absence of element number two does not cause disorientation, but simply absence of orientation, or ignorance of one's relation in space—e. g., a child, before it has learned through the different senses its relations in space. The importance of element number two will be apparent from the consideration of a few examples.

When a person with eyes closed, seated upright in a swing, feet off the ground, is rotated about the long axis of the trunk in the direction of the hands of a watch, there is a primary horizontal nystagmus in the direction in which the subject is actually moving. If the swing be allowed to slow down gradually, there will come a time when the nystagmus becomes reversed (secondary nystagmus)—i. e., horizontal against the hands of the watch. At this moment, if the eyes be kept closed and the feet off the ground, the subject will believe his body has reversed its direction and is now turning against the watch—i. e., in the opposite direction to that in which it may be actually turning. If the feet be put on the ground the eyes still being closed, the subject at once is disillusioned. The conditions of this experiment are so unusual that there is little or no background against which to interpret the incoming sensory impressions. The only experience the normal individual has of impressions from violent horizontal nystagmus is that associated with active turning of his body about the long axis. In the experiment, this plus the impressions from the nystagmus constitutes element number two, and the testimony of all the remaining senses, under the circumstances, offers no conflict, hence there is no disorientation, but merely an illusion.

One more example: An individual, absorbed in thought, standing on a ferryboat before the latter is loosed from its moorings, allows his eyes to rest on the water streaming past. Presently he believes the boat is moving. Here the background consists largely of a state of expectation that his boat is about to move, and the testimony of the senses, so long as his eyes see only the moving water, offering no conflict, an illusion is begotten—viz., that the boat is moving. A new psychologic background is then formed, based on the illusory motion of the boat. Now, when the eyes turn from the moving water and rest on stationary objects—e. g., on shore—the testimony of the senses—i. e., element number one—comes into conflict, not with the testimony of the other senses, as so many authors claim, but with element number two, in which at that moment was a false conception—viz., that the boat was moving. A revulsion of feeling follows, coupled with uncertainty and insecurity, which we call disorientation.

Examples might be multiplied indefinitely, but enough has been said to show that the psychologic element or background is a fundamental factor in orientation, and that disorientation does not consist in conflict between the immediate testimony of the senses, but between the latter and element number two, or the background derived from education and experience.

Dr. Jacobi wished to make a contribution from his own experience, for digestion by the specialists. He himself is subject to one particular kind of vertigo, which he always experiences whenever he is on the second story of a building, looking out of a window for just a few seconds. There is very little vertigo in his head, but a great deal in his feet and legs; they begin to tingle, and the feeling creeps up over the knees. A little cerebral vertigo is connected with the sensation. He would be very glad to have this explained, and explained soon, for he is believed to be an old man, and would like to be able to understand it soon. All his life he has been perfectly healthy, so far as he knows—he has had no brain disease, no tuberculosis, no syphilis—but just fifty years ago he had a typhus. It might be that he had something in his brain that gives him that peculiar vertigo or unsteadiness when he looks out of a window. He would like to know what it means, for nothing of the kind had been mentioned this evening, and it might be that in only this respect he was quite unique. He is sure he had it when he was thirty-five years of age. He does not remember having experienced it before his typhus, but he has had it ever since.

Dr. Duel thanked the men who had taken part in the discussion for their very able support of what he had felt to be a very desultory discussion upon a vast subject. The paper would have had but little value without the discussion.

The subject was one of much interest to otologists, and indeed to all whose attention had been directed to it. A few years ago Bárány had brought out certain functional tests whereby symptoms of an upset in the static state could be interpreted clinically with great exactness.

The psychic side of the subject was a most interesting field, and the discussion of it might well occupy an entire evening. No doubt, every one present had dreamed over it a great deal, but he himself did not wish to consume the time by adding any more dreams to the problem.

In talking with the President, Dr. James, over the best method of presenting this rather far-reaching topic before a general meeting of the Academy of Medicine, his first idea had been to present a series of cases which would illustrate the vertigo and other symptoms resulting from many different lesions, both peripheral and central; but he had found that in presenting the data it would be necessary to give in detail the results of a number of highly specialized functional tests, which even the specialists followed with great difficulty. For those who were not familiar with these tests an intelligible explanation would have required more time than was available.

Dr. James had, however, in the course of this preliminary talk, brought out a most interesting and ingenious phase of the subject which had not been mentioned during the discussion, and perhaps he would say a few words about it.

Dr. James spoke briefly of the result of posture upon the circulatory system.

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLOGY.

Meeting of January 14, 1916.

Acute Labyrinthine Irritation Due to Traumatism (Faulty Paracentesis).

Dr. J. H. Guentzer: This report may not be of interest to the otologist, but is here put on record to emphasize a protest made by the writer on several occasions against the advice of some otologists to the general practitioner—i. e., to incise the drum membrane of every acutely inflamed middle ear, for the purpose of drainage and to forestall further pathologic involvement. This is good advice to the family doctor who is well acquainted with the minute anatomy of the middle ear, who has good illumination and a clear field; but it is dangerous advice to the practitioner who is not well grounded in middle ear anatomy and is poorly equipped for illumination. While some would hardly dignify a myringotomy as an operation, it is well to remember that carelessly executed it has led to fatalities. Hemorrhages from the wounding of adjacent blood vessels, injuries that resulted in permanent deafness, and the wounding and infecting of the labyrinth, resulting in meningitis and death, have been reported in this connection.

On May 31, 1915, the writer was called by the family physician to see M. K., a male, thirty-one years of age. The patient was lying in bed on his left side, and had been suffering from pain in the right ear for two days; no discharge. Since the previous evening he had been unable to walk.

Examination of the right ear showed the membrana tympani to be slightly reddened; no bulging; a pin-head perforation about the center of the superior posterior quadrant, with a droplet of serous red discharge exuding. The patient had a spontaneous horizontal nystagmus to the left and rotatory on looking up; on attempting to rise he fell toward the right. The hearing was not impaired; there was no mastoid tenderness; no temperature; pulse, eighty-eight. A diagnosis of labyrinthine irritation was made, but no assignable cause could be elicited. Calomel and sodium bromid were recommended. rather as a placebo. This condition lasted ten days before the patient was able to leave his bed. At a subsequent office visit the patient confided the fact that his family physician was called the evening before the writer's first visit. The patient was told that he had an abscess in his ear, and the doctor used an instrument to open it, when immediately the patient became dizzy, nauseated, vomited, and was unable to walk.

The labyrinthine irritation was most likely due to disturbance of the footplate of the stapes. Fortunately, the middle ear and labyrinth were not infected, there was no discharge, and the patient escaped the dire consequences that might have followed such an infection.

Sigmoid Sinuitis With Abducens Paralysis in Acute Mastoiditis—Recovery Without Sinus Operation.

Dr. J. H. Guentzer: This case is of interest on account of its mild general course, with little evidence of sepsis in the temperature sequence, in spite of the fact that a sigmoid sinuitis was found on operation; and also on account of its abducens paralysis of the eye opposite to the involved mastoid without the usual clinical manifestations of sepsis, which rather substantiates the fact brought forward by Dr. W. H. Haskin, in his scholarly anatomic studies, that venous pressure on the sixth nerve involving its sympathetic fibers as it pierces the dura with the inferior petrosal sinus is responsible for the abducens paralysis. It is probable that an aseptic thrombus of the inferior petrosal sinus causes by pressure an edematous perineuritis of the sixth nerve, inhibiting its function. The absence of sepsis in this case seems to refute the opposing theory that such an ocular paralysis is caused by a toxemia.

The history in brief is as follows: On May 11, 1915, the writer called to see F. C., a boy, ten years old. The patient had pain in the right ear for one week, with no discharge. Examination showed the membrana tympani to be red and bulging, the mastoid quite tender, and the temperature 104°. A myringotomy was at once done under chloroform, given by Dr. Quinlan, the family physician. The patient ran a slight temperature, never exceeding 100°, during the next five days, with a moderate amount of pus discharge, but no abatement of the mastoid tenderness.

At the United Hospital, Port Chester, on May 16, 1915, a mastoidectomy was performed under ether. Pus and granulations were found in the mastoid cells and antrum. On removal of the necrotic area covering the sigmoid sinus, it was found to be of a decided yellow color, like a wax bean, and pulsating. The sinus was freely uncovered in both directions from the knee, and was found to be of the same abnormal appearance. A pus culture taken from the antrum at the time of operation was later reported by Dr. J. G. Dwyer as streptococcus mucosus capsulatus. Sinus exploration at this time was desisted from, partly on account of the fair general condition of the patient, as well as for the want of surgical assistance.

The urine examination was negative, and a blood count before operation gave a leucocytosis of 16,000, seventy per cent polynuclears, and twenty-nine per cent lymphocytes. Facilities for blood culture were not available. A convergent squint, diplopia, and a paralysis of the external rectus of the left eye came on the fifth day after operation. The ocular fundus was normal. Some anxiety was felt on account of the streptococcus mucosus capsulatus infection, but no further complications occurred.

Excepting for a rise in temperature to 103 3/5° on the seventh postoperative day, intestinal in origin, the case progressed in the usual manner, and the patient left the hospital after two weeks. After another month the entire mastoid wound was healed and the ear was dry. The left eye was again normal in three weeks.

DISCUSSION.

Dr. Friesner asked if he was correct in understanding Dr. Guentzer to say that the hearing in the first case was unimpaired, and he wished to know whether any tests were made with the noise apparatus; he presumed that no functional tests of the static labyrinth were made during the height of the condition.

Dr. Loughran did not think that one should consider he had made the hearing test properly until the sound apparatus had been used on both sides.

He then cited the case of a medical student who had tried to operate on his brother's ear, under local anesthesia. Just as he was making the incision the patient jumped the wrong way, and the knife was forced into the cochlea, and his hearing was destroyed. This incident brought to the minds of the students the fact that a myringotomy is a major and not a minor operation.

Dr. Friesner said that in Dr. Phillips' service at the Manhattan Eye, Ear and Throat Hospital there is a case very much like the one Dr. Guentzer had reported, excepting that no exploratory operation had been performed on the sinus. The patient was a young woman of twenty-two years. Dr. Phillips had performed a simple mastoid operation upon her left mastoid two weeks after an attack of acute otitis media. For ten days after the operation she ran an irregular temperature, with slow rises and falls, ranging between 100.5° to 102°. At the end of ten days, when he first saw her at the invitation of Dr. Phillips, she had some headache. She did not seem to be particularly sick, but her neck was a little rigid and the cervical vertebræ a little tender; beside that, she had an ankle clonus of the left leg. The fundus was normal. At this time she had a sharp rise of temperature to 104°, and Dr. Friesner did a lumbar puncture. The fluid was normal. Twenty-four hours after the lumbar puncture she had a temperature of 105°. Another lumbar puncture was then performed, for she was having severe headache and her neck was rigid. The fluid was again normal. A number of blood cultures were made and proved negative. Several blood counts showed 12,000 leucocytes, and seventy-five to seventy-six per cent of polynuclears. Dr. Phillips had almost determined to explore the sinus, when she suddenly became better; her headache disappeared as if by magic, and her temperature became almost normal; she now eats and sleeps well, and her neck is no longer rigid. Within the last three days, however, she has developed a sixth nerve paralysis on the left side. Of course, the diagnosis lies between a basilar process, a meningitis and a sinus thrombosis, or a phlebitis if not a thrombosis. The picture from the beginning, although she showed some of the symptoms of meningitis, was really not that of meningitis. She was not so sick as meningitis cases usually are, and the cerebrospinal fluid, of course, was negative. It would seem as though she had a phlebitis. In her present condition, however, Dr. Phillips' has concluded not to interfere.

Dr. Guentzer, answering Dr. Friesner's query, said that the case was seen in the country. He had been asked by the country physician to look over the patient, and had no opportunity to prepare to make any particular ear tests. His hearing was good with the usual tests of closing one ear at a time.

In the second case, the boy was never really very sick, but went through the entire illness with a very good appearance, and was fairly well, although he was combating a severe infection. He had these different signs and symptoms, and yet no high temperature or appearance of septic illness.

Complete Paralysis of the Cochlear Division of the Auditory Nerve, Associated With Normal Vestibular Reaction.

Dr. Hugh B. Blackwell: A. G., a well developed and apparently healthy woman, twenty-seven years of age, was admitted to the clinic of Dr. Duel at the Manhattan Eye, Ear and Throat Hospital, on November 17, 1915, with the following history:

Six years ago the right ear commenced to discharge, and has been running ever since. This discharge has been unattended by any constitutional disturbance. Five years ago the patient suddenly lost her hearing in the left ear. This loss of hearing occurred during the night, while she was asleep, and was unattended by any dizziness, nausea, or difficulty in equilibration. Since this attack the hearing in the left ear has not improved, and the patient has been compelled to rely entirely upon the right or discharging ear for audition. She has borne three healthy children, and both she and her husband deny specific infection.

Aural Examination.—Right ear: Canal contains foul pus, large perforation in membrana tympani, granulation tissue in fundus. Left ear: Canal dry, membrana tympani intact, no signs of previous suppuration, normal luster. Inflation shows left tube to be open.

Functional Examination.—Right ear: Patient can hear a loud whisper and low voice. Left ear: With the Bárány apparatus in the right ear, patient is unable to hear the loudest noise. Weber referred to right ear.

The left, or deaf, ear was then irrigated with cold water. In thirty seconds, marked nystagmus with dizziness and sensation of nausea was produced on looking toward right. The left ear after a half hour interval was irrigated with hot water, and the nystagmus was reversed, producing also dizziness and sensation of nausea. The duration of this after-nystagmus was about a minute and a half. The patient was then whirled in a revolving chair ten times in ten seconds, in alternate directions. After each whirling there was produced an after-nystagmus upon looking in the opposite direction from which she was turned, the duration of which was about thirty seconds.

On November 4th, four days after these tests were made, they were repeated, with substantially the same results.

On the day of the patient's admission to the clinic, she was sent to the laboratory and her blood was taken for a Wassermann examination; it was pronounced negative. She was then placed upon a provocative specific treatment of iodid and mercury protoiodid, and at the end of a week her blood was again taken for Wassermann examination and again pronounced negative.

Conclusions.—Some authors believe that the function of the vestibular nerve can be destroyed and at the same time the cochlear division of the eighth nerve remain unparalyzed. Theoretically, there is no reason why the reverse of this proposition should not occur also—namely, complete paralysis of the cochlear branch, the vestibular nerve escaping. However, in a cursory review of the literature, he had not been able to find any cases reported in which the cochlear had been totally paralyzed and the vestibular branch entirely escaping. The case is interesting in that it shows the pathologic independence of these two divisions of the eighth nerve.

DISCUSSION.

Dr. Dixon stated that he would not recommend the use of either potassium iodid or mercury as a provocative. Mercury will cause a negative Wassermann in some positive cases.

Dr. Braun said that it was a very unusual and interesting case. The sudden onset of the condition made it probable that it was due to a hemorrhage. Whether the hemorrhage occurred into the labyrinth or into the region of the nucleii of the cochlear nerve in the medulla is very hard to say. It is difficult to imagine a hemorrhage occurring into the cochlea which would not also involve the vestibular structures. It is more probable that it was a small hemorrhage involving the nucleus or root of the cochlear nerve.

Dr. Guttman asked if Dr. Blackwell had examined the spinal fluid. This might have shown a positive reaction. It was quite a well known fact that the cochlear branch of the eighth nerve is more sensitive than the vestibular branch; the cochlear branch, therefore, may be destroyed, yet leave the vestibular branch unaffected. The reverse, however, is very rare—e. g., that the vestibular branch should become destroyed and leave the cochlear branch unaffected.

Dr. Dougherty said he wished to emphasize the fact that a negative Wassermann signified nothing either way.

Dr. Blackwell, in closing, said he was inclined to think that the lesion was in the peripheral apparatus rather than in the central nuclei. The patient had no dizziness on the following day, and no nausea. It was difficult for him to conceive of a central lesion occurring so suddenly without irritation of the vestibular nerve. Of course, it was impossible to secure salvarsan for provocative treatment last November. Suppurative diseases of the ear frequently attack slowly the semicircular canal system, and adhesions and walling off processes occur which theoretically would protect the cochlea, but in almost all of these cases the cochlea became affected.

Streptococcus Mucosus Capsulatus Infection of the Mastoid Bone.

Dr. Robert Loughran: Of the various pathogenic organisms producing purulent infections of the middle ear and mastoid process, the streptococcus mucosus capsulatus has come to be the one that most arouses interest, not only on account of its tendency to produce a very rapid general involvement of the entire bony structure of the mastoid, but also because of its tendency to develop "a most dangerous and insidious latent period which may justly occasion apprehension concerning the outcome of any inflammatory ear disease which has its origin in the activities of this infective agent."

In the opinion of many writers, it is a bacterium closely associated with pneumococcus, yet with sufficient characteristics individual to itself in its growth in various media, its reaction to immunologic and fixation experiments, and its pathologic reaction in diseased tissues, to entitle it to especial attention. All admit that much work still remains to be done in order to place it in the position of knowledge to which its virulence entitles it.

Buerger has divided the pneumococci into four groups—typical forms, small forms, large forms, bacillary forms—the third of which "may easily be mistaken for the streptococcus mucosus capsulatus, but are, as a rule, very definitely lancet-shaped, whereas the latter are possessed of a more rounded or biscuit-shaped form." His classification of streptococci is: (A) 1. Streptococci without capsules. 2. Streptococci with capsules. (B) Streptococci with mucoid capsules, which he describes as the streptococcus mucosus capsulatus, differentiating them carefully.

On the other hand, Hanes, in an "Immunologic Study of the Pneumococcus Mucosus," concludes that the organism described by Schotmuller "represents a well defined group with characteristics which indicate a close relationship to the pneumococci rather than to the streptococci"; and that the name "pneumococcus mucosus" should be adopted for this group, rather than "streptococcus mucosus." He further notes that in this series of cases of lobar pneumonia caused by the pneumococcus mucosus, sixty-six per cent had died, and that when the organism had been found in the blood, all had died.

Lyell concludes that the type reactions for the pneumococcus mucosus group are the same as for the true pneumococci; and it is interesting to note that the majority of the strains he studied (ten in all) were isolated from cases of acute mastoiditis.

Dochez and Avery present tables showing the relative occurrence of organisms of the different types during the year 1912-1913, and record the fact that in thirteen per cent of their cases the organism fell in the group classified as the pneumococcus mucosus, and that in 1913-1914, eight per cent were of the mucosus group.

While there is uncertainty among bacteriologists as to just where the organism should be placed in the classification of bacteria, there is no doubt of its virulence and insidious tendency for the production of late complications. For the purpose of recording the various symptoms and conditions which the streptococcus mucosus capsulatus may produce, three cases were cited at length, the reader of the paper concluding: that in meeting this infection experience has taught that we are dealing with an organism whose power of rapid and extensive destruction of the mastoid bone may easily be able to combat the best efforts against it, and that in order to conserve the patient's best interests, early and frequent bacteriologic examinations should be made of all discharges in suppurative otitis media, in order to be in position to take advantage of every point that could be of value in determining the necessity for operative procedure; and, further, that having made an attempt to stop its destructive course by operation as soon as there is any indication of mastoid involvement, there is still an uncertainty of prognosis to consider, even after the wound is healed and the patient apparently is well.

DISCUSSION.

Dr. Dixon said that the views expressed in Dr. Loughran's paper corresponded with his own ideas in regard to the streptococcus mucosus capsulatus. At the New York Eye and Ear Infirmary it was the routine practice to examine smears in every ear case, and his experience during the last ten or eleven years had led him to the conclusion that the mucosus capsulatus occurred in about five per cent of all cases. Since he first began to study this germ (a good many years ago) he has been impressed with its extreme virulence, and has acquired great respect for it. As Dr. Loughran had said, it is not possible to determine when the end has been reached. To illustrate the length of time which such a case will run, Dr. Dixon read the history of a case which he had reported in a paper read before the Medical Association of Greater New York in February, 1913, and which was published in the Annals of Otology, Rhinology and Laryngology of the following June. The total duration of this case was from May 24th to the 12th of December, when it terminated fatally. During this time the patient was under constant observation, and had four operations. Dr. Dixon said that of course this was an unusual case, but that they have many cases at the infirmary which go out apparently well, remain so for a month or six weeks, and then come back with meningitis. Two or three years ago Dr. Whiting had a private case, an adult male, who was in rather poor condition (leucocytes only 4200). He did fairly well and left the hospital not long after his mastoid operation and returned to his home in Long Island, being dressed at Dr. Whiting's office for about a month; then on a Thursday he came in, and was progressing splendidly. On the following Saturday night he had a chill, was readmitted to the infirmary, and on Tuesday he died of meningitis. Such cases are not at all uncommon.

Dr. Dixon said he had been harping on this subject for a great many years. The "hurry up" case reported by Dr. Loughran was rather an unusual one, and he had not personally seen or known of such a case. It was a very rapid one. As a rule, these cases at the infirmary progress exactly as any other dangerous infection, but the trouble with them is that a point is reached where the germ apparently becomes latent, and there are no symptoms, except possibly a little sagging of the canal and the discharge. Some time ago he had been called in consultation by Dr. Adams to see a patient, sixty-three years of age, without temperature, pain or tenderness, and advised operation; but two advised against it. The patient asked if it would do any harm to wait a day, and Dr. Dixon replied that he thought not. The next day Dr. Adams operated and found dura exposed to the extent of three-eighths of an inch. The patient recovered. There may be no symptoms, no pain, no temperature—nothing but the discharge and the mucosus capsulatus. Cases of this nature demand at least an exploratory operation, if the duration has been over two weeks, for the patient's life is in increasing danger with each succeeding day.

Dr. Page agreed with Dr. Dixon, that it was not safe, as a rule, to watch these smouldering cases with a streptococcus mucosus infection longer than two weeks. He had never regretted operating on such cases.

Dr. Guentzer said that some two years ago he had a streptococcus mucosus capsulatus case occurring in a little girl of eleven, and had reported it extensively in a discussion at the opening meeting of the section a year ago. The point that was particularly interesting was that the child had a mastoiditis in May, and went through the entire summer until late in September, and then died. She had a sinus thrombosis and jugular involvement of the right ear, and a metastasis two weeks later in the ankle joint, which went on to necrosis of the astragulus. A few weeks after that there was another metastasis in the hip joint and femur, then in two of the ribs and shoulder blade, then in the hip joint of the other side, involving the pelvic bones. There was not a single spinous process of the back which escaped involvement; the skin and muscle from the occiput to the buttocks had sloughed away; the child also had an abducens paralysis of the opposite side. One of the men at the consultation said he had never seen a worse infection, and it was certainly the most typical one that he himself had ever seen.

Dr. Dixon wished to know if any of the members present had seen or heard of a case of recovery from meningitis caused by streptococcus mucosus capsulatus.

Dr. Braun said that he had seen a case, two or three years ago, with Dr. McCullagh, at the Beth David Hospital. He thought that Dr. McCullagh had reported the case at one of the section meetings. The patient was a girl, about twenty-five years of age, who had had a simple mastoid operation done. A week later she developed a hemiplegia of the opposite side, strabismus, rigidity of the neck, and a Kernig, and was very apathetic. She continually tore at her bandages, and was very hard to manage.

A lumbar puncture showed cloudy cerebrospinal fluid which contained large numbers of streptococcus mucosus capsulatus. It was deemed inadvisable to operate, as the case was thought hopeless. However, she improved, and in several weeks was entirely well.

Dr. Blackwell said he had understood Dr. Loughran to state that he would operate on these cases at the first sign of mastoiditis. He could not agree with Dr. Loughran on that point. He had operated upon a number of these cases at the infirmary, and had seen a number of them get well without operation. One must remember in dealing with the capsulatus infection, as with all other infections, that there are two important factors to be considered, both of which are subject to wide variations in intensity: first, the resistance of the patient toward infection; second, the virulence of the infective agent. The variations in amount and intensity of these two factors have been responsible for the number of recoveries from mastoiditis caused by the capsulatus infection occurring in his own experience.

Dr. Hays asked whether any one knew of cases where the symptoms became less on account of the virulence of the organism becoming attenuated. Many of these cases seem to lose their acute symptoms and go on to a quiescent stage, and he had wondered whether it was because the germ loses its virulence, or whether it continues as strong as in the beginning.

Dr. Loughran, in closing, said that the germ loses its virulence only in dry secretions, and that the virulence could be restored very promptly by putting it in a proper medium. Every man has to be guided mainly by his own experience. His own experience with this organism had made him very much afraid of it. He had seen one case of it in his own famiy, and could quote two other very interesting cases, one of them being in a small boy whom he had seen for Dr. McKernon, the other one occurring in a man who had an acute earache at six o'clock in the morning. Dr. McKernon saw him in the afternoon, and he then had an acute exudative otitis. The next morning the boy had a mastoiditis, and an appointment was made for operation that afternoon. The attending physician objected—that being his general attitude toward all surgical procedures. The boy was operated upon the second day, and had an immense cavity. He finally got well after eleven or twelve weeks. Had he been operated upon the first afternoon there would not have had to be such a large operation nor such a prolonged convalescence. In another case the patient was awakened at three o'clock in the morning with an earache. Early in the morning the drum ruptured. He began immediately to have signs of mastoiditis—a little fullness in the upper posterior portion of the drum and periosteitis in the canal, and suddenly developed a moderate mastoid tenderness.

New Instruments: An Improved Method for Draining the Tympanic Cavity in Purulent Otitis Media.*

By John Guttman, M. D.

DISCUSSION.

Dr. Loughran asked if there was complete healing of the drum in every instance. There is always danger of contraction later, with permanent perforation. It was interesting to know that Dr. Guttman makes a good sized hole.

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*See page 389.

Dr. Heller asked whether Dr. Guttman administered an anesthetic each time; for if not, he did not understand how the operation could be accomplished satisfactorily in every instance, especially with young children, for even a lancing causes a good deal of pain, and such a measure as that proposed requires even more time, and the patient would suffer more. He also took exception to Dr. Guttman's method of blowing out the eustachian tube with the catheter after opening the drum. That did not seem to be a wise procedure when the middle ear was filled with infected material. Such matter gets into the antrum readily enough without being forced there.

Dr. Hays said that Dr. Guttman had come to the infirmary one afternoon and operated upon about half a dozen cases by this method, most of them under local anesthesia. That in itself is almost as painful as opening the drum. So far as the puncture itself was concerned, the cases were all acute cases, and Dr. Guttman claims that the instrument is chiefly for use in chronic cases.

Dr. Hays said further that his chief objection to the instrument in its present form was that unless it was very sharp, one was apt to make the incision half or a third what was desired, and leave a little piece which would hang in and block up the opening. He had suggested to Dr. Guttman that interchangeable points be made so that one could always have a sharp one. In acute otitis where such excellent results are obtained with the knife, it hardly seems necessary to get out a special instrument; but in cases where the incision has a tendency to diminish in size or close very rapidly, it is more than worth while to make a good sized opening such as this instrument accomplishes.

Dr. Page said that in his experience he had seen little good result from repeated myringotomies. If after one, or at most two, free incisions the swelling and bulging of the drum membrane persisted, he had seen no marked change in its appearance affected by a third or fourth incision to promote better drainage. Usually the bulging in such cases was caused by swollen mucous membrane and granulations, and he thought a large percentage of them finally came to mastoid operation in spite of oft repeated myringotomies, whether performed with a knife or trephine. Personally he did not feel that the trephine offered any great advantage over a knife of fair size.

DR. FRIESNER said that in applying this trephine to the drum membrane, the contents of the tympanic cavity can never be under such pressure as to offer sufficient resistance to cause the instrument to cut through the drum. Wherever the instrument is efficacious, the resistance necessary to cut or punch out the circle must come from the inner tympanic cavity, and there certainly must be some danger of injury, if it be only the mucoperiosteum, or possibly even the bulb. This problem had been put up to a mechanical genius a number of years ago, and he made the statement that no drum membrane, no matter how much fluid or pus the middle ear contained, would be under such tension that one could, by a circular punch or trephine, cut out the drum membrane clean without the resistance of the inner tympanic wall behind it.

DR. SCRUTON considered that there might be some difficulty in keeping the delicate trephine in good cutting order; he also believed a more or less prolonged experience in the use of the instrument would be required to develop an ability to manipulate it successfully in every instance. He stated that in his opinion the opening made by the trephine would give a free and uninterrupted drainage which cannot always be maintained by use of the myringotomy knife. The instrument presented by Dr. Guttman is worthy a trial by every otologist. It may prove a decided advance over the present myringotomy knife.

DR. GUTTMAN said he was very glad the instrument had brought out so much discussion. All he could say was that he had used it upon more than thirty cases, and none of them had showed an ill effect from it. Some of these cases had been destined to operation for mastoiditis by different surgeons, yet this operation having been performed and proper drainage established, the cases got well.

Replying to Dr. Loughran's query about a permanent opening: he had expected this objection, and for this purpose had brought two cases for observation, one of them being a colleague, operated upon a year and a half ago, who shows no sign of the operation in his drum membrane, and the other a case that had been operated upon more recently. In not a single instance did the opening remain permanently.

As regards anesthesia: he had used mostly local anesthesia, applying three or four drops of a four per cent cocain solution in the upper wall of the canal. In some cases that worked marvelously, and the operation was absolutely painless; in other cases there was some pain. The operation had been performed even on some children of eight or nine years, under local anesthesia; they cried a little, but the operation took only a second or two longer than an ordinary paracentesis. A few cases had been operated upon under nitrous oxid, but in most instances, especially with adults, local anesthesia was employed, and five minutes later the operation was performed with hardly any pain in some instances and very little in others; as the operation requires so little time there was no real suffering in any case.

The paracentesis knife is surely quicker, as it is thrust in the drum membrane and the thing is done; but it may have to be repeated many times on account of the insufficient drainage. With this trephine one gets a drainage that could not be secured with any knife whatsoever. Most of the cases operated upon by this method had been acute cases, and the results were excellent. The colleague referred to was absolutely well in twelve days and had no more pain, and being a physician, he was well qualified to judge the result.

Dr. Friesner had claimed that there was not enough rigidity in the drum membrane and not enough resistance to secure a good opening. That was pure theory. Dr. Guttman said that he had performed thirty-odd cases, and got through the drum membrane in every instance with the greatest ease, and never had any bad results. Many acute and most subacute or chronic cases will get good drainage by this method.

Dr. Heller said that he had found local anesthesia very unsatisfactory in incising the drum membrane. Lately he had been employing somnoform, which gives a very short general anesthesia—lasting only a minute; and with a child it is really ideal. It takes half a minute to a minute to get the patient under the effects, and one has fully a minute for the work. There is no vomiting or other ill effects. Dr. Halstead had published an article in the *Laryngoscope* (August, 1915, p. 562), stating that he had had a great deal of experience with it and no bad results.

Dr. Page inquired whether somnoform is ordinary ethyl chlorid.

Dr. Heller said that somnoform is ordinary ethyl chlorid eighty-three per cent, ethyl bromid one per cent, and the rest is methyl chlorid sixteen per cent.

Dr. Loughran asked if there was any danger point.

Dr. Heller replied that, practically, there was not. It is given until the patient is under its effect, and then stopped; a one cubic centimeter phial is enough for an ordinary anesthesia. One can determine the condition by the breathing. The patient is at first a little excited, and then goes off into a deep sleep. No assistance is required.

Dr. Guttman said that Dr. Heller had stated there was no danger to be feared from somnoform. It was invented by a French dentist, but there had been some fatal cases following its use, so one should not be too confident with it, for there is some danger connected with it. Nitrous oxid seems the safest and most sure anesthetic.

Dr. Guentzer said that he, too, wished to sound the same warning. Some ten years ago it had been tried at the Manhattan Eye, Ear and Throat Hospital for tonsil and adenoid cases, and a few of them did not do well. It is not so simple and safe as Dr. Heller seemed to think.

Dr. Heller replied that he, too, had tried it on tonsil and adenoid cases, and it was not at all satisfactory there. The dental supply man said that he knew of men who were using it, and came to the hospital to demonstrate it on two cases, and it was absolutely useless. It is not suitable for anything except very short operations. The effect is too evanescent for anything like a tonsil operation.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Regular Meeting, Held November 16, 1915.

DR. GEORGE W. BOOT, THE PRESIDENT, IN THE CHAIR.

Case of Hypophyseal Disease.

DR. OTTO J. STEIN exhibited a patient upon whom he had operated almost a year ago by the transnasalsphenoidal route. The patient was in apparently good health up to four years ago, when she first noticed a reduction in vision in the left eye, which was soon followed by attacks of headache, becoming more severe and bitemporal in character. Associated with the headache was a vertigo, which later became so severe that there were four or five attacks daily, completely incapacitating the patient from any work whatsoever. After a year and a half the vision in the left eye began to rapidly decline, and then evidence of impairment in the right eye manifested itself. The patient came under the observation of Dr. George Suker about a year ago, and from a careful examination of her eyes, and with the skiagraphic findings (as could be readily seen from the plates shown), a diagnosis of hypophyseal involvement was made, and the patient presented to Dr. Stein for operation, which was performed about a year ago. At the time of operation the vision was practically nil, and she was obliged to have someone lead her about. This, with the severe headaches and vertigo, added materially to her suffering. The pupils were equal and reacted to light and accommodation. There was no extraocular muscular involvement. The left eye was totally blind, only slight light perception being present, the disc being choked, and evidence of neuritis present. The right eye showed a choked disc of three to four millimeters; no atrophy as yet, but there was a neuritis. No nystagmus.

The symptoms, from a pituitary gland standpoint, were as follows: Patient, thirty-three years of age; married; no children; menstruation began at thirteen and ceased at twenty-three; adiposity—weight being two hundred and forty-five pounds; skin dry; scanty hair; polyuria; functionless genital organs, and some infantilism. There was no sugar in the urine. Wassermann negative. Blood pressure one hundred and twenty-five pounds systolate, ninety diastolate. Gastro-intestinal tract negative. No motor paralysis. No Rhomberg. Tendon reflexes slightly reduced. From these symptoms it can be readily seen that we had to deal with a case of pituitary adiposity, with a typical Fröhlich's syndrome.

The Hirsch technic was followed in operating—namely, a typical submucous resection of the posterior portion of the nasal septum was done, with the addition of removal of the right middle turbinate. This gave sufficient access to the sphenoid region. Great care is necessary in elevating the periosteum from the anterior face of the sphenoid, as it is essential to maintain an unbroken membrane throughout, in order to thoroughly protect one's retreat, and thus avoid the possible danger of infection of the meninges from the nose.

After entering the cranial cavity by removal of the floor of the sella, the gland readily presented, and after determining somewhat its character by exploring with a probe, and concluding that it most likely was an adenoma, he was satisfied to limit operative measures to a decompression.

After six days the patient made a rapid convalescence, and recovered quickly from the symptoms previously presented, and is today entirely free from headaches or vertigo, and vision has returned to normal in the right eye, and so far improved in the left that she performs all the duties of her household, including sewing, reading and writing, etc., without being conscious in the least of any deficiency in sight.

It is interesting to note, in this connection, that the rhinologist has at his hand a technic for performing an operation in this region that is so simple, and withal so satisfactory, and at the same time is free from the objections resulting from the methods employed by the general surgeon. On inspection of the patient's nose, one could never detect the slightest evidence of her having any former nasal operation, and the patient suffers no inconvenience from such a method of operation.

Case of Rhinophyma.

Dr. Stein said this case commenced in the same way that many such cases commence. This condition usually occurs in those who are exposed a great deal to the open air in all kinds of weather—that is, in engineers, firemen, conductors and motormen, drivers and teamsters. There is frequently an element of force back of it. This man noticed it after blowing his nose violently, when something seemed to give way. He felt a dilatation of the vessels. He had a slight rhinitis at the time, just as many of these cases have. After this he noticed the enlargement. Then the lobulated masses began to form. The nose feels soft.

In looking at this nose, one thinks of several conditions. One is likely, however, to think more of rhinoscleroma until you touch the nose. Then you notice the difference. This condition is nothing but a hyperplasia of the skin.

Probably the best exposition of rhinophyma has been given by Jacobi, of Freiburg, in his Atlas, but, like all dermatologists, he attributes all of them to some form of acne. This has been denied by rhinologists. In the case shown, there was no history of acne, and no evidence of it about the man now. Dermatologists claim that there is a hyperplasia of normal skin, but particularly of those parts in which there are sebaceous glands. The condition starts with a hyperemia, which disappears easily on pressure in the first stage. In the second stage there are enlarged vessels and veins, and, finally, these small masses of tissue, which they say is nothing but hyperplasia from the sebaceous glands, later forming lobulated masses, until they deform the nose and pull it down, as shown in this case. The third stage is much rarer than the other two, which are quite common.

Dr. Stein did not think this particular type has ever been recognized in women, which is another interesting point.

On looking inside the nose, it could be seen that the man was entirely free from any involvement of the interior of the nose.

Carcinoma of the Antrum.

Dr. George W. Boot presented a patient with carcinoma of the antrum. He lost his eye four years ago from an accident, and before he had any symptoms of cancer. He came to Cook County Hospital three months ago, complaining of right-sided nasal obstruction, and wanted something done to relieve his breathing. There was a large polypoid mass present, which the speaker removed through the nose. The carcinomatous process evidently began in the antrum. A Denker operation was then planned to relieve the nasal obstruction, a radical operation being out of the question, since the growth extended across the median line in the roof of the mouth. When the external wall of the antrum was exposed, it was found destroyed to such an extent that a curette could easily be introduced. The interior of the antrum was thoroughly curetted out, the internal and external walls and much of the floor of the antrum being removed. The patient then left the hospital, but returned a day or two ago in the condition presented. The cheek is much thickened with the growth. The hard palate is much infiltrated, and the infiltration extends almost across the roof of the mouth. In the speaker's opinion, the growth is absolutely inoperable. He is receiving Coley's serum and is being treated by the X-ray, but with little hope of accomplishing any permanent good.

DISCUSSION ON DR. STEIN'S CASE.

Dr. Joseph C. Beck, referring to the case of rhinophyma, recalled a case which he and Dr. Friedberg had seen, which was even more extensive than the one presented. He thought it might be interesting to tell what they did in this case in the way of treatment. The patient had a tumor that hung down over the mouth. He was a man about sixty-five years of age. He was operated, with excellent result. The operation consisted of a decortication of the whole area of the nose involved; then some skin from each side of the nose and face was brought together to cover the defect. It made a pinched nostril, but the result has been satisfactory, and there has been no recurrence. The growth was removed right down to the cartilage. Kaposi has reported this as necessary in these cases, so that no vascular and verrucous structure will remain.

Dr. Beck thought the result in Dr. Stein's hypophyseal case was excellent, and he, as well as the patient, was to be congratulated.

Dr. Stein, in closing the discussion, said that he intended to perform the decortication operation in the case of rhinophyma, as he understands that is the only method of treating these cases with any result. Dr. Beck said that in his case he took off all the skin down to the cartilage. Dr. Stein would think that a faulty thing to do; because you have to depend upon skin formation. You should leave a little layer of skin there for it to properly epidermize. If you take it all off, you get scar tissue contraction, unless you do a skin grafting, or draw the skin from the neighboring parts over the raw surface. If you are going to allow it to heal without any grafting whatsoever, it is a mistake to cut so deep. You have got to carve it out, as a sculptor would carve, with a razor, and dissect all around the cartilage, but always leave a little skin underneath for healing. With this method, he is led to believe that the results are very good.

DR. BECK said he was afraid he had been misunderstood. Unless you remove the growth down to the perichondrium, there is a recurrence, and the nose must be immediately covered. He did not wish it understood that he left the nose exposed or did any skin grafting whatsoever, but made a dissection from the side of the face, in the form of flaps, and brought them together over the tip of the nose, so that the nose was covered with a healthy skin. To carve out this tissue and leave some of the skin would prove futile, according to authorities.

Paper: Suspension Laryngoscopy, With Report and Presentation of Cases.*

BY JOSEPH C. BECK, M. D.

DISCUSSION.

DR. S. A. FRIEDBERG thought that certain points should be emphasized in connection with the use of the suspension apparatus. The first concerns that of technic. When the instrument is introduced the tooth-holder and spatula are very close together. This prevents satisfactory illumination of the lower part of the pharynx and epigottic region. If care is not used in manipulation, one may get an edema of the epiglottis and aryepiglottic folds; or if too long a spatula is used there is the possibility of it going back of the arytenoids into the mouth of the esophagus. Any unnecessary or excessive manipulation in the supraglottic region is very apt to cause an edema. The speaker has found this to be so, particularly in children with multiple papilloma of the larynx, in using the simple direct method in operation. This brings up the question that if we are to use this instrument in children, particularly in cases of multiple papillomata, whether or not it is advisable to do a preliminary tracheotomy.

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*See page 330.

Dr. Beck referred to the difficulty experienced in suspending some cases. This reminded the speaker of a case which both he and Dr. Beck had tried to suspend at the County Hospital a few days previously. In this case there was a diffuse papilloma, involving one cord, with aphonia of a number of months' duration. There was complete anesthesia, but it was impossible to keep the spatula in place on account of the gagging reflex. After several attempts they were obliged to desist. It was interesting to note that immediately following the suspension operation efforts the respirations went up to about thirty-six a minute. The patient was very dyspneic; pulse about one hundred and sixty; the girl could not breathe very well lying down, and had to sit up in bed. A sedative was given, and the next day she was in very fair condition. On looking into the larynx the next day some evidences of traumatism were to be seen. There was considerable swelling of the vocal cords, with whitish exudate over the false cord, showing that the tip of the spatula must have come up over the vocal cords, producing this traumatism. The larynx was also deeply injected.

Dr. J. Holinger said that Dr. Beck, in his paper, spoke of suspension laryngoscopy in patients suffering from asthma. Dr. Holinger wished to draw the essayist's attention to a series of articles on the treatment of asthma by means of this method. The attacks stopped at once, but the cocain was always given the credit and not the laryngostomy. The cocainization of the trachea, at the bifurcation, will stop even very bad cases of chronic bronchial asthma for one to many years. After that time, the same procedure may be repeated, with the same result. Patients who had gone through terrible trials with bronchial asthma have been kept in absolutely comfortable condition as long as the observation lasted.

Dr. Beck asked if only one application was necessary.

Dr. Holinger said yes—just one application at a time. If necessary, after years a second treatment could be given, but these cases are the exceptions. The exposition of the matter referred to could be found in the *Correspondenzblatt für Schweizer Aerzte*, two or three years back.

Dr. S. A. Friedberg wished to add one more point to his remarks, namely, in regard to the use of anesthesia in these cases. In any operation about the respiratory tract the use of prolonged anesthesia is dangerous. That has been found out especially by those doing bronchoscopic work. The speaker had one death on the table, while removing a foreign body, after about an hour. The anesthesia in this case was given by an expert. Jackson and others have recognized the possibly serious dangers in working about the respiratory tract, and Jackson rarely uses general anesthesia in children in doing foreign body work, and very seldom uses cocain. The speaker, also, in the last few years very seldom uses cocain. That is a danger that should also be recognized. In using an excessive amount of cocain we are apt to have a dangerous complication, as Dr. Ingals has pointed out.

Dr. Beck has asked the speaker why he does not employ this method in foreign body work. He has not felt the necessity, because it may be managed so easily the other way. At least, that has been his experience, and he believes Jackson inclines to the same view. One objection Dr. Beck himself brought out, namely: In the case of unruly children who are to be suspended, an anesthetic must be given in order to accomplish anything. In passing the bronchoscope, without the use of suspension, this is not necessary.

Dr. George W. Boot wanted to call attention to two points in the report. First, amputation of the epiglottis for tuberculosis or other conditions is very easy under suspension laryngoscopy. Second, in cases where edema of the larynx is feared following suspension laryngoscopy, it is advisable to do intubation as a precautionary measure. The speaker has seen papilloma of the larynx cured by wearing the intubation tube alone. It ought to be still more efficient after removal of the papilloma.

Dr. Beck, in closing the discussion, showed another case, in order to illustrate how well he could speak. Speech is the essential thing in these cases. When this patient consulted him he was very hoarse. He had a fibroma on the cord, which was a good-sized growth, not as large as a pea, but just about that form. It dropped subglottically and would not come up readily. It looked like a little polyp. It was situated anteriorly. This growth was removed by the suspension method described.

Dr. Beck wished to make a strong plea, based upon his experience, for the use of this method for small growths, in accurate removal.

Dr. Boot spoke of intubation, and that is what the speaker had wished to say in answer to Dr. Friedberg's remarks. He has done that after removal of papillomas in children, so as to avoid the possibility of edema. He likes to use tracheotomy in papillomas in children for a longer period, because it prevents their recurrence, to some extent, and is considered good treatment.

The morphin is an excellent thing in these nervous patients, and hyoscin, which the speaker used, as well as scopolamin. If Dr. Beck were to try suspension again on the patient referred to by Dr. Friedberg, he would give her morphin and atropin or hyoscin to get her quiet. He has found that this inability to suspend some cases is more of a mental condition. He did not see any exudates, but the fact that there was some trauma in this case should not deter anyone from the use of the method in other cases. The thing to watch out for is the trauma of the arytenoid, by too long a spatula. He was glad Dr. Friedberg brought out that point. We must thank Albrecht and Killian for using the extension on their spatulas for epiglottis. By using a little shorter spatula of the Lynch apparatus than the one exhibited and carefully avoiding the arytenoids, the operation is easier, and the view better.

Dr. Beck did not know about the articles spoken of by Dr. Holinger, namely, the treatment and cure of asthma by one application of cocain. That is worth while trying. He had a case at the present time of a man who has such severe attacks of asthma that it is pitiful to look at him in one. Adrenalin and cocain locally have never done him any good.

Dr. Holinger asked if he injected the cocain down to the bifurcation.

Dr. Beck answered that he did not.

Paper: A Suggestion Regarding the Rinne Test.*

BY ROBERT SONNENSCHEIN, M. D.

DISCUSSION.

DR. ALFRED LEWY wished to add a suggestion to those made by Dr. Sonnenschein in regard to the technic of the Rinné test, with reference to the reading of the results. He thinks the expressions "R. plus" and "R. minus" alone are most meaningless. A great deal of confusion has been caused by the use of expressions plus or minus, without indicating at the same time how much the air conduction and how much the bone conduction was relatively lengthened or shortened. We expect to find a minus or negative Rinné in a conduction deafness, but this is only relatively true. We find it in a severe conduction deafness, not in a mild one. On the other hand, it is very common to find a negative Rinné, but a bone and air conduction both shortened, but, nevertheless, minus Rinné, in severe cases of nerve deafness. The speaker believes that if we would indicate the result of our Rinné test by an equation showing the bone and the air conduction, and the normal for the fork used, a great deal of confusion would be eliminated from the literature.

DR. GEORGE E. SHAMBAUGH was pleased to listen to this discussion of the functional hearing tests, especially because this important part of otologic work is the one most frequently gone over carelessly. An experienced otologist is often able to make a tentative diagnosis with a good deal of accuracy from the history the patient gives. But a positive diagnosis of the type and degree of deafness can only be determined by carefully carried out functional tests. Taken alone, the Rinné test is much more valuable than either a Weber or the Schwabach test, but even the Rinné test cannot be relied upon by itself to make a diagnosis in all cases. It is important here, just as in much of our work, to have a regular method of procedure, and to run through a number of the more important functional tests in every case where the diagnosis is in doubt. A positive or a negative Rinné is often a relative affair, and it is only by studying this test in connection with the others that an accurate conclusion can be reached regarding the cause of the deafness. A shortened positive Rinné may have the same significance as a negative Rinné, and in advanced unilateral nerve deafness a negative Rinné on the affected side has the same significance as a positive Rinné does in most cases.

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*See page 455.

Dr. J. Holinger said that the international scheme for recording Rinné's test accepted that each part of the test should be noted separately. For example, strike the fork and note the bone conduction; then strike the fork again and note the air conduction; then draw the difference. This was done with the same idea that Dr. Sonnenschein brought out. But just there is where Bezold brings up the point that we do not need absolute numbers; we need only the difference of hearing by air and by bone conduction in the same ear for our diagnosis. To get that difference we have to strike the fork only once. Therefore, the question of hard or easy striking is avoided. If you have a certain amount of routine in the test, you will easily reach the point where you can make four or five tests in succession on the same ear, and not vary more than one or two seconds in your findings.

The argument of making original investigation may be accepted only conditionally. Dr. Shambaugh says that we have to stick to a certain routine for statistic purposes. The speaker would add that any time-consuming additions to this routine ought to be cautioned against.

Dr. George W. Boot thought functional testing of the ear is apt to be slighted. Several years ago he was in a clinic in Philadelphia where they had one tuning fork (laughter). In another clinic they had three or four forks and an old-fashioned Galton whistle. In one New York clinic they had a set of Bezold tuning forks, but did not use them.

In this connection he wished to call attention to the little book by Sonntag and Wolff on "Functionspruefung des Ohres." It not only gives the methods of making the tuning fork tests, but the tests of the vestibular system as well, and all in very compact form.

Dr. Sonnenschein, in closing the discussion, replying to Dr. Lewy and Dr. Shambaugh, said there are seven different kinds of Rinné, depending on the cases in which either the air or bone conduction are lengthened or shortened. As stated in the paper, he did not discuss this point, but simply desired to call attention to the fact that the actual air conduction, simply from the scientific standpoint, as a matter of pure knowledge, is much longer than that ordinarily assumed by most writers. It is necessary to remember, as Dr. Shambaugh so well pointed out, in making any diagnosis, where using tuning forks—which are practically all subjective tests—that you are entirely dependent upon the statements of the patient. There is no objective method of measuring the acuity of hearing. It, therefore, is necessary to take a view of all the tests, as well as the other clinical data, in forming a diagnosis.

In answer to Dr. Holinger, he would say that he has on his cards the whole international formula, as decided upon at the Buda Pesth Congress in 1909, and he puts down the figures on these cards.

As far as stating that all you desire for the Rinné test is the difference between the bone conduction as determined on the mastoid, and then the air conduction, he agreed with Dr. Holinger, and stated that this ratio is essentially determined in the ordinary routine. It simply seems to him a desirable thing to know accurately how long by air conduction the tuning fork is heard in most cases. Has not our knowledge of hearing by air conduction taken on a different viewpoint since the introduction of the monochord? It was always assumed that high tones are best heard by air conduction, whereas, as a matter of fact, with the monochord it has been shown that by bone conduction the very highest tones are heard much better than by air conduction. Therefore, if we can find anything after a long series of observations which tells us anything more definite, it may be important in later on arriving at some conclusion regarding functional testing. So far as its requiring more time, the speaker was willing to grant that, but it took only a minute longer. That point he had emphasized in his paper. If there is anything in the method described, after examining several thousand cases, we will know it; at any rate, nothing can be lost by performing the test.

ABSTRACTS FROM CURRENT LITERATURE.

EAR.

The Streptococcus Mucosus in Its Prognostic Relation to Acute Suppurative Disease of the Middle Ear.

WINCKLER (*Archiv. für Ohrenheilkunde,* Vol. 96, p. 193). The consideration of this important subject from the clinical standpoint, which is so much at variance with the usually held views, deserves to be read in full. The author in the period from 1907 to 1912 saw only one case where the pus escaping through the paracentesis wound showed the streptococcus mucosus, as compared with fifteen cases of infection through the streptococcus pyogenes, and four with the streptococcus lanceolatus.

This case was a child of six years, where healing took place in eight days. There has never been any relapse. Four weeks after the attack the streptococcus mucosus was found in the tonsils when removed. Shortly before this a nine-year-old brother was seized with acute middle ear disease which showed streptococcus lanceolatus. This case required opening of the mastoid within twenty-four hours.

These experiences have caused the author to entirely give up the examination of the pus obtained by paracentesis as of no practical value, inasmuch as the child affected with the streptococcus mucosus made a prompt convalescence without mastoid involvement, while the brother, who showed streptococcus lanceolatus, immediately came to mastoid operation.

During the years 1907 to 1914 Winckler performed one hundred and nineteen operations for acute mastoiditis. In nineteen cases occurring in patients from sixteen to sixty-four years, streptococcus mucosus was found once in pure culture in a woman of sixty years, as compared with thirteen pure cultures of streptococcus, one of lanceolatus, one of staphylococcus pyogenes aureus, and two with staphylococcus albus. One patient suffering from infection with streptococcus albus and two with streptococcus pyogenes, the former forty-five years old and the latter fifty-eight and sixty-four years, showed all the characteristic symptoms ascribed to a mucosus infection—absence of pain, absence of fever, characteristic appearance of the drum membrane, small quantity of serous secretion after the paracentesis, eight to ten days later healed middle ear; six to ten weeks later a relapse with complaint of headache and slight tenderness over the mastoid, which up to that time had been lacking.

The X-ray picture in all these cases at that time showed cloudiness of the bone, and the mastoid operations which followed revealed extensive disease extending into the cranial cavity with involvement of the dura. The older the patient the less variation there will be in all types of infection. A typical clinical picture with the streptococcus mucosus has no value, when found in a person of advanced years, inasmuch as other forms of infection can show the same picture.

As regards acute otitis in children, the author found that in fifty-seven streptococcus infections, twenty showed the streptococcus lanceolatus, five the staphylococcus pyogenes aureus, two the streptococcus albus, one the bacterium coli, thirteen mixed infections, and only in one, in a child of a year and a half, where there was a double-sided acute mastoiditis, was the mucosus found in pure culture.

Among the thirteen mixed infections there was one case of a child of three years, where the streptococcus pyogenes and mucosus were found together, and another case in a child of eleven years, a double-sided mastoiditis, where the streptococcus lanceolatus and the mucosus were found together. The case of pure mucosus infection showed a sudden onset with high fever, spontaneous perforation of both drums, rapidly increasing swelling, first behind one ear and then the other. A complete mastoid operation produced a prompt healing on one side. On the other side a radical was necessary. Both cases made prompt recovery, so that in four to five days they were discharged from the hospital.

As regards the rôle of the mucosus in mixed infection the author is in doubt. He is inclined to feel, however, that its presence is not of particular importance. The author's views in regard to the involvement of the mastoid in very severe acute otitis are at variance with those of most writers. They are based upon the study of the X-ray pictures in such cases. In many severe cases with profuse discharge the X-ray shows the mastoid cells perfectly clear, not only at the beginning, but also at the end of the inflammation. In other cases at the very onset there is a decided differenec to be noted betwen the diseased and healthy mastoid. It is interesting to remark that so far he has not been able to note the disappearance of the cloudiness from the mastoid after the cure of the disease.

The importance of the X-ray in all acute cases is dwelt upon, although it is admitted that it is necessary that the mastoid should be pneumatic as well as the disease one sided, in order to permit of its use. Where the usual methods of examination fail in arriving at a definite diagnosis, much importance can be derived from a picture showing distinct cloudiness in the sinus region or one where it is shown extending into the pyramid. Such cases, in spite of an apparent favorable convalescence as regards the middle ear infection and of the sinus, of mastoid involvement, edema, pain, sensitiveness, etc., are to be regarded with caution. The cloudiness can indicate permanent changes in the mucous membrane or reparatory changes in the diseased bone. It can, also, however, mean an encapsulated focus of inflammation, deep seated, in the region of the sigmoid sinus, which requires only a new exciting cause to become active.

The examination of the bone removed from the patient of sixty years, suffering from pure mucosus infection, showed fibrous transformation of the medulla, containing chiefly lymphocytes with a few leucocytes; in other words, a chronic process with acute exacerbations. The histologic examination of the bone in the patient suffering from staphylococcus albus infection and with streptococcus infection, gave exactly the same picture. This was equally true of the microscopic findings in the bone of the child of a year and a half, with double sided mucosus infection, and of the case of mixed infection in the child of three years.

The prognosis in middle ear affection, where the involvement of the mastoid has been proven, always demands the bearing in mind of a possible complication. Where there is a favorable condition as regards the size of the middle ear and of the aditus, the infection can go on to complete healing. On the other hand, in many cases it is undoubtedly true that after years the foci of disease still remain, in spite of the fact that the disease in the middle ear has promptly healed. In such cases one of two possibilities for further trouble is to be borne in mind. The diseased process advances in an insidious way from the foci that remain in the interior of the bone. Here they play an important rôle in the intracranial complications which develop. Indeed, they are of more importance than that usually ascribed to cholesteatoma. Second, the foci of disease which remain can become incapsulated and a healing produced or a quiescent state. Later the diseased area is lighted up by an acute cold or an inflammation of the tonsil, and any of the well known complications can develop.

Often in these cases the hearing is normal and the drum membrane intact, especially in young individuals. In children where the bone has been removed, evidences are found to show that the process was by no means an acute one, but rather pronouncedly chronic.

The importance of the embryonic tissue found in nurslings has not been definitely determined. The author agrees with Wittmarck that the middle ear inflammation may serve to prevent the transformation of the embryonic tissue in the early months of life, and in that way lead to middle ear and mastoid infection. He does not, however, agree with Wittmarck in his contention that in such infections of the mastoid in children there is little risk of complication except where the streptococcus mucosus is present. It is his feeling that there must be, in addition to the pneumatic form of the temporal bone, a wide-open middle ear and auditory canal, in order to insure a favorable outcome of the inflammation.

In all the cases of extensive disturbance of the mastoid where a pronounced pneumatization was present, he found a small antrum and narrowness of the middle ear. This is to be explained by the mechanic interference with drainage produced by the pronounced swelling of the mucous membrane in a small middle ear cavity. The infection of the streptococcus mucosus is then, as far as the author's cases are concerned, much less common than it would appear from the statistics of other operators. In one hundred and eight cases of tonsil enucleation the streptococcus mucosus was found only twice. Whether the infection with this organism has a characteristic peculiar to the region, or whether it can have only a temporary significance, he cannot determine. His statistics are to be compared with those of Mark, who found in his cases of mastoiditis four and one-tenth per cent of streptococcus mucosus infection, as compared with Stütz, where it was present in thirteen per cent of the cases.

The pathologic institute at Bremen, where all the histologic examinations were made, is firmly of the opinion that the mucosus infection gives changes in no way different to that produced by other organisms. *Harris.*

The Influence of Vasomotor Disturbances in Childhood Upon the Organ of Hearing.

STEIN AND POLLAK (*Archiv. für Ohrenheilkunde*, Vol. 96, p. 216). After an exhaustive analysis of this subject the authors reach the following conclusions:

Vasomotor irritability in childhood gives rise, in addition to disturbances in various organs, to trouble with especial frequency in the organ of hearing. The clinical symptoms, as far as the ear is concerned, are the result, without question, of disturbances in the circulation in the organ of hearing, especially as the result of anemia in the blood vessels supplying the ear, particularly those in the region of the auditory nerve itself.

They are both of a subjective and objective character. The subjective symptoms are noises in the ear and painful sensations; the objective, fatigue symptoms involving the acousticus, the function of the cochlea, and in some cases of the vestibular apparatus.

As proof of the direct relation between the vasomotor disturbances elsewhere and those in the ear, are to be mentioned, first, the fact that evidences of disease involving the ear almost always show themselves in the symptom complex of the characteristic clinical picture of cerebrovasomotor disturbances (headache, vertigo, brain fatigue, etc.); and second, the fact that the effect on the function of the inner ear is always proportionate to the intensity of the vasomotor disturbances.

The question whether vasomotor disturbances can also give rise to organic changes in the auditory nerve can without hesitation be answered in the affirmative. In such cases all factors which can give rise to an abnormal function of the vasomotor nerves—infectious disease, physical and psychical trauma, disturbances of equilibrium—give rise to or at least contribute to the development of the disease in the inner ear.

In the treatment the shutting out of all factors which may injure the vasomotor systems, proper diet, careful hygiene, and especially control of all influences arising from the school, are of prime importance.

Especial emphasis is to be laid upon the fact that by the careful observation of disturbance in the organ of hearing as the result of vasomotor irritability, trouble in childhood can be recognized at an early age, the further development of which in later years must, under all circumstances, be feared. Attention at the proper time can check the development of the disease, or at least put a halt to its rapid development.

Harris.

The After-Treatment of Brain Abscess, With Special Reference to Abscess of the Cerebellum.

BUELLER (*Archiv. für Ohren-, Nahren- und Kahlkopfheilkunde,* Vol. 98, p. 58), in an exhaustive way, considers all the various controversial questions in regard to the immediate and remote treatment of brain abscess. He reviews the teachings of various operators, and then gives the conclusions of the procedure in the Erlangen clinic.

In regard to a large or small incision of the dura, he advises, on account of possible infection of the arytenoid space, an incision which is not too long. The temperature following the operation is apt to be elevated for the first few days, whereas the pulse frequency rapidly decreases. Vomiting is of frequent occurrence after the operation. Its significance often gives trouble. It may be due only to an irritation of the stomach, and on this account need not always be considered as an unfavorable symptom.

The changes in the eyegrounds are often to be observed for several weeks after the operation. Indeed, there may be an increase of the optic neuritis. They have no significance, however, from a prognostic standpoint. The nystagmus, as a rule, decreases after the opening of the abscess, but returns with increased force in case there is a retention to the discharge of the pus.

A particularly alarming symptom is the paralysis of breathing. This may occur several days after the operation. The explanation is to be found in increased brain pressure, although too tight a packing can be accountable for it. No way is known to prevent this complication or to combat it when it occurs.

A prolapse of the brain is one of the most serious complications met with. It is dependent upon increased intracranial pressure and infection of the brain content. The most careful drainage, even by lumbar puncture when necessary, is essential to avoid this. Authorities do not agree whether a large or small incision is more conducive to brain prolapse. In the treatment, large incisions are of value. A broad opening conduces to much better drainage, and in that way the retention of secretion is avoided. For small prolapses a permanent bandage is sufficient. Large prolapses call for the removal of tissue where it is edematous and much infiltrated. The liability of a further prolapse after the incision demands careful consideration, however, before doing so. Great weight is to be laid upon the general treatment after the operation. All congestion of the head which may cause an increased blood pressure is to be avoided.

The position of the patient is also of importance. He is apt of himself to lie upon the diseased side, for especially in cerebellar abscess there are disturbances of equilibrium which give the patient a sensation of falling toward the diseased side. Neumann makes the patient lie on the side corresponding to the direction of the nystagmus.

In regard to when the first dressing should be done, the author advises, if there are no special indications demanding, that it be done before, and that it be changed on the second or third day. The frequency of the dressings depends upon the individual case. Care of the discharge from the wound is very important. Henke believes that in all uncomplicated brain abscesses which result fatally, the cause is to be found in faulty drainage. Adhesions of the walls from the fibrin deposited are not infrequent. These of themselves can cause retention of the infected material.

The author dwells upon the importance of bearing in mind the possibility of a second abscess to explain the persistence of the symptoms complained of. They are similar to those produced by the retention, and a differential diagnosis must be made between the two. The existence of multiple abscesses is denied by many authorities. Their etiology could be ascribed to a metastatic origin; pyemia, especially, can cause their recurrence.

The choice between gauze packing and drainage tubes is gone into at length. Authorities in favor of each are quoted, Politzer, among others, making use in his clinic of iodoform gauze packing impregnated with perhydrol, five to fifty. In the Erlangen clinic a glass or rubber drainage tube is used when there is profuse suppuration, and gauze packing when the discharge is only slight. Iodoform gauze is generally employed.

The position of the tube is of importance, an unfavorable position leading usually to retention. When symptoms of retention arise, the permeability of the tube should be at once tested. Some authorities are in favor of removing the lining membrane of the abscess. Curetting of the abscess wall should be undertaken only in a most careful manner. The author leaves the wall entirely alone.

Boric acid powder and iodoform can be used in the abscess cavity upon occasion. If there is much odor due to anaerobic bacteria, a solution of peroxid of hydrogen can be used. Neumann is especially in favor of it.

For purposes of exploration of the abscess cavity, to determine its extent and direction, Ruttin and Neumann, among others, recommend digital examination. This is strongly condemned by other surgeons. Killian's nasal speculum has been used for this purpose. Mention is made of Whiting's encephaloscope for this purpose, but apparently the author has never used it. The Killian bronchoscope has been employed for the same purpose with success.

The routine use of irrigation is no longer to be recommended. In the proper cases and in the proper manner, however, it is not dangerous, and often of great value. In the Erlangen clinic it is regularly used. The all important point in irrigation is to employ as little force as possible. For this purpose a middle ear canula with a rubber ball is employed. The wound is then carefully dried and a fine boric acid powder blown in.

When in the treatment for symptoms of retention it is necessary to make a brain puncture, a number of instruments can be employed, the needle, scalpel, or forcep. None of these is without possible harm. Which is best to use depends upon the particular case. The needle has the disadvantage over the knife, in that the pus mingled with the necrotic brain tissue can easily stop it up. The author uses first the canula. If this does not succeed, he employs a knife. The danger of an exploratory incision in the brain must be constantly borne in mind. Pathogenic bacteria can be easily introduced into the brain. The most scrupulous care and sterilization of all instruments employed is, therefore, necessary.

Encephalitis and meningitis are the two complications of brain abscess to be borne in mind. In more than fifty per cent of the fatal cases, meningitis was the contributing cause. The recognition of meningitis is often very difficult, on account of the necessity of differentiating from the symptoms due to the abscess. Only less dangerous than meningitis is encephalitis. It can follow directly after the operation, and be accountable for the paralysis of breathing.

The duration of the after-treatment is very variable. Politzer states from three to six weeks. In the author's three cases that resulted in a cure, the time was forty-five, fifty-eight and sixty-one days, respectively. The prognosis, in the author's opinion, is not to be regarded as altogether favorable. Indeed, even in cases reported as cured, Heimann feels that an interval of several years must elapse before this can be properly claimed, for often after a considerable interval complications and relapses take place. In a general way it may be stated that the prognosis depends upon the character of the complication, the size of the abscess, and, above all, upon the drainage conditions. One should be very careful in any case in making a prognosis. Death can arise from the abscess itself, if proper drainage is not secured and maintained, or it can come from the extension of the process into the brain.

The author concludes his paper by giving the histories of the six cases recently operated upon in the Erlangen clinic.

Harris.

LARYNX.

Thermotherapy in Certain Forms of Catarrhal and Tubercular Laryngitis. (Preliminary Report.)

MOURE AND GOT (*Revue de Laryngologie,* May 15, 1916). The method is that of Bier, applied to the larynx. It previously has been used in Germany in Killian's clinic by Albrecht, who made use of heated boxes.

Got has devised an electric heater so arranged that it can be connected with the street current. Further, it is so constructed that the temperature shall not mount beyond a certain degree, and so burns of the skin are avoided. A thermometer is provided for measuring the temperature of the skin.

The treatments are given preferably daily, and last from three-quarters of an hour to an hour.

The authors report their results in a series of thirty cases, and draw the following conclusions from their investigations:

1. It gives excellent results in cases of spasmodic laryngitis during treatment, but the benefit does not last beyond the conclusion of the treatment.

2. In acute cases of laryngitis thermotherapy causes a rapid disappearance, usually at the second, third or fourth treatment, of the troublesome symptoms, and diminishes to a considerable degree the length of the disease. In grip the cure is obtained less promptly than in simple forms of laryngitis.

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XXVII.

THE DIAGNOSIS AND MANAGEMENT OF VASOMOTOR DISTURBANCES OF THE UPPER AIR PASSAGES.*

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It has been known for some years that an individual sensitized to a given proteid may exhibit a characteristic reaction if the proteid is brought in a soluble form in sufficient concentration into contact with a scratch of the skin. In the present connection it is of importance for us to recognize and interpret correctly the significance of this skin reaction. I shall, therefore, briefly describe it and its various degrees of intensity.

The proteid to be tested is applied to a scratch on the skin of the arm. After five to fifteen minutes the positive reactions are indicated by varying degrees of local disturbance. These disturbances of the skin may be ranged in order of intensity as follows: In some cases the first perceptible alteration consists in a sharply circumscribed white area, not elevated, bordering the scratch for a distance of one to three millimeters. We may find in other individuals the first manifestation to consist of a slightly reddened raised area. In more pronounced disturbances the area of swelling is more extensive, and is more or less white in color, being surrounded by an area of reddening of varying size. When this degree of disturbance appears, it is usually accompanied by itching. It may, in marked cases, attain a considerable size, the edematous area reaching two to three centimeters or more in diameter and surrounded by a centimeter or more of hyperemia.

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*Read before the American Laryngological Association, May 9, 1916, at Washington, D. C.

In the course of thirty minutes, or at most one hour, the reaction fades, and only the slight trauma occasioned by the scratch is left. In the case of certain proteids there may appear on the following day an alteration at the site of the scratch characterized by appearances of inflammation rather than of angioneurotic disturbance. The skin bordering the scratch becomes reddened, slightly elevated and firm. In the course of another twenty-four hours a trace of suppuration may supervene. These disturbances disappear in two or three days. It was at first thought that these inflammatory manifestations were the result of a chance infection, but they have occurred so regularly after testing with certain proteids as to suggest a definite relationship to the proteid substance itself. They follow most frequently tests with staphylococcus p. aureus and albus and streptococcus pyogenes. Among the foods cocoa is apt to show it.

While the occurrence of the skin reaction may be regarded as indicative of sensitization to the proteid employed, yet it is premature to say that a direct relationship invariably exists. Certain observations, for instance, made in the course of hay fever immunization, which I have elsewhere recorded, show that there is not always a skin reaction proportionate to the intensity of the nasal and ocular symptoms. These are matters requiring further study.

During the past two years over four hundred cases showing vasomotor disturbances of the upper air passages were examined by the skin test with different proteids, for the purpose of determining, if possible, the diagnostic value of this reaction, and ascertaining its relationship to the clinical manifestations. For many of these disturbances the special exciting proteid was indicated by the patient's history, as in cases of hay fever, intolerance to fish, horses, egg, etc., but exclusive of the hay fever patients, the larger number gave a clinical history in which the causative agent was unknown to the patient.

Cases of vasomotor rhinitis of recognizable reflex origin, and asthma from cardiac and renal disease are not included. In many instances, particularly of nasal disturbance, the diagnosis of a reflex cause, such as from the genital organs, is difficult to establish, but wherever possible a physical examination from an internist or the family physician was obtained.

CLASSIFICATION OF PROTEIDS.

Recent studies have established a phylogenetic classification of animals and plants, based upon the serum reactions. As the result of the studies, chiefly of Nuttall for animals and of Metz and Gohlke for plants, we are enabled to construct a phylogenetic tree for both kingdoms. The application of these studies to our subject is considerably simplified by our ability to eliminate many members of the animal and plant kingdoms which have no direct relation to man.

Animals.—The primitive proteid molecule in the animal kingdom represented by protozoa exhibits a differentiation in the direction of the molluscs or shell-fish, and passing upward gives off the branch of the crustacea. Above this are the vertebrates, with the branches respectively of fishes, birds, the main trunk terminating in the mammals. In the present connection the mammalian orders which concern us are represented by the domestic animals, and by those which serve as foods, or whose skins as articles of clothing.

Plants.—The phylogenetic tree of the plant kingdom proceeds from the bacteria upward, and divides into the monocotyledons and dicotyledons, the first being represented chiefly by the large family of grasses and by the orders of iris and lily. The trunk of the dicotyledons terminates in the order Compositæ, or family to which the ragweed belongs, and gives off along its course a large number of orders, which have been shown to be of anaphylactic significance, the most important members of these being the nuts, the legumes and the rose family.

PREPARATION OF MATERIAL.

It is of importance to have the test material in the form in which it exists when exciting symptoms. Consequently, we should have it either in the raw or cooked state, respectively, or in both, as the case may be.

It should also be in a stable form, and as pure and concentrated as possible.

This statement is especially emphasized by the fact that we find that different persons vary widely in their degree of sensitization to a given proteid; consequently, one person may react to a proteid dilution of a given strength, while another individual, sensitized to the same proteid, may require a hundred or more times greater strength of the solution to elicit an equivalent skin reaction.

In the case of animals, preparations should be made both of those parts capable of entering the respiratory mucous membrane, as flying scales or hairs, and also of the serum, and of those parts ordinarily ingested, such as egg and milk. Extracts from the epidermal structures are easily obtained by placing scrapings from the skin, hairs or feathers, respectively, in twelve per cent alcohol. Sterilization is accomplished after a few days by the action of the alcohol, and the suspended particles or clear filtrate may then be used. The material may be tested for its sterility, but in no case have I found it contaminated or even after months show signs of decomposition. These extracts appear to keep without deterioration, and I have now two made from horse epidermis, which have been kept at room temperature for over a year, and are apparently as active as when first prepared.

In the case of animal foods, crude but satisfactory material is readily prepared by expressing the juices in a meat press, and subjecting them to rapid desiccation by a blower.

The same method may be followed in the case of plant material. Oils may be removed by leaching with carbon tetrachlorid. Both these animal and plant extracts may then be preserved when thoroughly dry without deterioration.

The proteids of egg and milk can be obtained commercially in the form of a pure powder, as egg albumen and casein, respectively.

While the refinement and separation of the different proteids present in a given material is desirable and represents a field of important research, yet the above mentioned relatively crude preparations will suffice to answer our first clinical question as to whether the individual is sensitized to the material in question.

In the present work cases were examined both with preparations made along the above lines by Dr. Turnbull in the throat department of the Massachusetts General Hospital, and also by pure refined proteids prepared by Mr. R. P. Wodehouse in the botanical laboratories of Harvard University.

PREPARATION OF BACTERIA.

Suspension of bacteria killed by coagulation of their albumen may be injected intradermally, and the presence or absence of a reaction noted on the following day, as already practiced for a number of diagnostic tests, but I have also found it possible to procure the bacterial proteids in a soluble form, capable of exciting an immediate skin reaction in the same manner as that obtained from the pollen and other plant and animal extracts. At the beginning of the work cultures were grown by Dr. C. G. Page, instructor in bacteriology at the Harvard Medical School, on agar slants, then they were exposed to ether vapor, the object sought being to kill the microorganisms without coagulation of their albuminous constituents. After remaining in ether vapor four days, the colonies were transferred to a twelve per cent dilution of alcohol with water, in order to extract, if possible, the proteids of the bacteria. This alcoholic dilution was chosen, as it had been found by the writer to preserve best the anaphylactogenic properties of pollen and keratin extracts. The quantity of bacteria was about one-third by volume that of the alcohol dilution added; so that, on centrifuging, the bacterial bodies occupied one-third and the fluid two-thirds of the tubes. After a week the fluids were tested on the skin of patients in the same manner as that employed for the diagnosis of plant sensitizations.

Since then a variety of methods of preparing bacteriologic material have been developed by Dr. A. E. Steele of the Massachusetts General Hospital, Dr. I. C. Walker at the Peter Bent Brigham Hospital, Dr. Cleaveland Floyd, and Mr. H. M. Baker at the Harvard Medical School, to all of whom I am greatly indebted for the opportunity which they have given me to test preparations made with this object in view.

In the case of plant pollen, I prefer to keep it in a dry form, as it will then keep unchanged for an indefinite period. I have flowering specimens of grasses and of ragweed in my herbarium, gathered over thirty years ago, the pollen of which still gives pronounced skin reactions in patients sensitized to these plants.

A method was evolved by Mr. Wodehouse for the extraction of ragweed pollen from the flower heads, which enables one to obtain it in a purer form than has hitherto been the case. Plants of ragweed are gathered in the flowering stage and the heads of the flowers stripped off. The habit of this plant is to open the flower heads in succession, and consequently at a given time some will be found to have discharged their pollen and others to be as yet unopened. After drying in air the heads of flowers were washed in carbon tetrachlorid to remove oils, etc., and it was found that most of the pollen came out in the process. Very little solid matter except the pollen will pass through the muslin used for straining. The pollen grains, having passed through, are allowed to dry and are seen to be somewhat paler than those obtained in the natural way, the difference in color being probably due to the removal of lecithin.

The same method is applicable to the other anemophilous plants which furnish a large amount of pollen.

We are thus enabled to obtain the pollen in a relatively pure form, and consequently can make solutions of known proteid strength. Furthermore, this material has not been subjected to the risk of change by treatment with proteid precipitants, and approximates closely to the original proteids in their natural state as they actually reach the exposed mucous membranes.

Experiments were undertaken in regard to preserving pollen and pollen extracts. As I have stated in a previous paper, a dilution of alcohol in water proved most serviceable. The important object to be attained is a solution which will check decomposition with the minimum loss of strength of the active principle. It was found that pollen extract in six per cent alcohol or less showed, after a few days, development of bacteria and proteid decomposition. A dilution of eight to ten per cent was sufficient to check putrefaction, and if the bottle was completely filled with the solution, it remained free from alteration. If, however, the bottle was frequently opened or left half full, there was a prompt invasion of the yeast organism, with development of vinous odor. This was later followed by the appearance of the mycoderma vini and general turbidity. Solutions from twelve to fifteen per cent could be left open without alteration, and there was no development in them of the mycoderma. These solutions were found to contain an unstable albumin and a relatively stable proteose. The former is readily coagulated by exposure to air, by treatment with alcohol of above twenty per cent, and by heating to sixty degrees C. The proteose, on the contrary, suffers but little on exposure to air, and will endure boiling without precipitation. This observation is in harmony with the familiar fact in wine making that at the close of the first fermentation a clear fluid is present containing the albuminoids of the grape in solution. These are in part thrown down at the first racking and consequent exposure to oxygen, rendering the wine turbid, and requiring often the admixture of isinglass to effect a clarification.

For this reason pollen solutions should be kept as far as possible from exposure to air, and if in the older ones a cloudiness occurs, it should not be filtered, as thereby the albumin would be removed, leaving only the proteose in solution.

The globulins of the pollen grain were separated by Mr. Wodehouse, but were found to be without anaphylactogenic properties.

METHOD OF ENTRANCE OF FOREIGN PROTEIDS INTO THE BODY.

Obviously, under natural conditions the mucous membranes of the respiratory and the intestinal tract afford ready means of access for these substances when inhaled or ingested, and we may consequently speak of such forms of allergy as (1) inhalation or (2) ingestion anaphylaxis.

The possible existence of a third form should be considered, namely, where the symptoms may arise from (3) autolysis of bacterial proteids when in contact with the mucous membranes. In a recent paper I have called attention to this possibility, and have shown that in certain cases of perennial vasomotor rhinitis and bronchitis it is possible to elicit an immediate skin reaction by the application of soluble bacterial proteids to a scratch. Since this paper was written further observations have tended to confirm these views. Such cases are dependent upon the more or less periodic invasion of the mucous membranes by the bacteria to which the individual is sensitized, while during the intervals of infection the patient remains free from vasomotor disturbance.

At least two other possible bacterial causes may exist. In the first instance it is theoretically possible that septic foci, as in chronic tonsillitis, with accumulation of lacunar débris, may be the source of sensitizing proteids; also it is conceivable that the intestinal canal may permit the development of bacteria which may undergo autolysis, and the consequent liberation of proteid material which is anaphylactic for the individual.

The above system of classification is a tentative one, and while for certain portions we may believe that we have sufficiently confirmatory evidence, we are not as yet in a position to accept it as a whole without further investigation.

In the present paper I have endeavored, with this classification as a basis, to study a series of vasomotor disturbances occurring in private practice and in the throat department of the Massachusetts General Hospital.

I.—INHALATION ANAPHYLAXIS.

Under this heading two main types are recognizable, the first being seasonal, the second perennial. In the first instance we find that the symptoms appear at a definite time of the year, and are represented by the very large class of hay fever patients, the symptoms coinciding with the flowering of the special plant in question. The second or perennial form occurs at any time of year whenever the exciting proteid enters the system through the respiratory tract, as in sneezing or asthma from the presence of horses and cats.

I (A)—SEASONAL INHALATION ANAPHYLAXIS OR HAY FEVER.

In this affection recognition of the causative agent is considerably simplified by keeping in mind the following facts: In the first place, our search should be chiefly directed to the monoecious or dioecious plants. As I have previously pointed out, it is those plants which distributed a large amount of pollen in the air which excite hay fever symptoms. Entomophilous plants which accomplish cross fertilization through the agency of insects are able to be much more economical in the production of pollen, and it is consequently not likely that such pollen can come in contact with exposed mucous surfaces under natural conditions. Such pollen, furthermore, is more viscid and sticky, the object evidently being to enable it to cling to the bodies of the insects which visit the plant in question. It is necessary, on the other hand, for anemophilous plants which depend for their fertilization upon the agency of air currents, to produce a large amount of pollen, and pollen, furthermore, which is relatively light and easily detached from the anthers.

Recognition of these facts enables us to determine with much more certainty the causes of hay fever in a given locality. We may, therefore, pass over the entomophilous plants and confine our attention to the anemophilous representatives. Such are, in this vicinity, certain trees, especially maples and oaks, the grass family as a whole, and certain species of the wormwood family. Although the pines and other gymnosperms produce a very large amount of pollen, yet I have not so far found any instance of hay fever being caused by them. It is in the next place necessary to consider the season of hay fever symptoms. If they occur previous to the flowering of the grasses we shall obviously have to look for the exciting cause in the early flowering shrubs and trees, and among these birch, maple and willow are chiefly responsible. About the middle or the last of May the oak begins to flower in this region, and tests should, therefore, be made for this family. The flowering period of these trees is relatively brief, and consequently the hay fever occasioned by them is less likely to disturb seriously the patient's mode of life.

In the case of grasses we have a flowering period extending from the middle of May in this locality until the first week in July, and the attack of hay fever which they may excite is not only more prolonged but more severe.

Examination of the Compositæ is very much simplified by recognizing that the proteid or active agent of all its members is essentially the same. While minor differences may exist, yet one active pollen will, for diagnostic purposes, cover the whole order. An interesting example of this is found in two of my patients who spend the winter in Cuba, and who there in February suffer severely from a plant known locally as Escoba amarga, used by the natives to keep fleas from their dwellings, which has been identified for me by Professor Oakes Ames as Parthenium hysterophorus Linn. After the return of these patients to Boston in the early spring, they are free from hay fever until the flowering of the grasses, when a moderately severe amount of vasomotor irritation appears and lasts until the first of July. They then remain comfortable until the middle of August, when severe hay fever appears and lasts until frost. Examination shows that these patients are sensitized to grasses and to ragweed, and that the Cuban plant excites marked skin reaction. Tests of this plant on numerous other sufferers from ragweed who have never been in Cuba excite marked skin reaction in all cases.

Tests, furthermore, of different members of the Compositæ, from the earliest flowering ones, such as coltsfoot, to the late blooming asters, show that with sufficiently strong solutions all ragweed cases will develop skin reactions. Under natural conditions, however, throughout the greater part of the United States, the chief exciting cause among the Compositæ is found in the ragweed, the structure of which will repay examination. It will be seen that the plant is characterized by the possession of long spikes containing the male flowers, and by the localization of the female flowers at the base of the spikes. The amount of pollen which this plant distributes is very large, and it is also extremely light. The flowers open in succession, beginning with the central spike, a few discharging daily—the scattering of the pollen being consequently maintained for a long period. Another plant of the Compositæ which resembles the ragweed in its method of flowering is the Baccharis halimifolia, a favorite plant in shrubberies, although its power of causing hay fever has not hitherto been recorded. The plants are dioecious, and the amount of pollen formed is considerable. The more showy members of the Compositæ, such as field daisy, goldenrod, asters and other garden representatives, possess a relatively small amount of pollen, as well as one of a more generally viscid character. Patients consequently more readily escape, unless the plants are brought into the house.

Examination of the present series of cases has led to the conclusion that the number of proteid types concerned in exciting symptoms is much smaller, at least for New England, than has previously been supposed. During the period preceding the flowering of the grasses, nine cases presented symptoms which terminated by the first or the middle of May, according to the seasons. These patients showed no sensitization to grasses, but reacted chiefly to the maple, birch and willow. Fifty-seven cases showed sensitization to grasses, and one hundred and eighty-three were positive to ragweed and other members of the Compositæ. Sensitization to members of the rose family alone was found in but one case, although a number were found in association with grass sensitization, where it was evident that grasses were the predominating factor.

With regard to the July or midseason forms of hay fever, I believe that with exception of the chestnut and possibly some garden flowers, the chief causes are found either in some special distribution or exposure to either late blooming grasses or early blooming Compositæ.

It is at this season also that the cases of olfactory vasomotor rhinitis or pseudo hay fever are most commonly found. In this affection the fragrance of certain heavily scented flowers excites vasomotor disturbances, ranging all the way from sneezing to actual asthmatic paroxysms. The action of pollen can be definitely excluded by wetting or removing the anthers without influencing the vasomotor symptoms. Skin tests, furthermore, of the pollen show no reaction. The phenomena are plainly reflex, and the path of transmission appears to be along the olfactory rather than the optic nerve. The flowers most commonly responsible are lilies, hyacinths, sweet peas and peonies. The condition occurs either alone, or in association with true hay fever, and is more common in neurotic or impressionable individuals, although not confined to such, several of my cases occurring in highly intelligent individuals, possessing no discoverable neurosis.

It may here be noted that during the hay fever attack a general hypersensitiveness of the patient is apt to appear towards plants for which at other seasons he shows only a minor or no reaction. One must, therefore, be on his guard at this time to observe carefully the relative preponderance or degree of development of the various skin reactions, and he will probably be safe in confining his therapeutic endeavors to the plant which causes the most marked disturbance.

I (B)—PERENNIAL INHALATION ANAPHYLAXIS.

(a) Of Plant Origin.—Under this heading I have included those cases of vasomotor disturbance in which the exciting proteid is derived from some other part of the plant than the pollen. Two cases of severe sensitization to flour were found in bakers, who, however, had no hay fever, and who did not react to wheat or grass pollen. These cases, furthermore, could eat bread without disturbance. One case of sensitization to potato was found in a cook. Eight cases of severe sneezing from orris root were encountered in women using face powder containing this as an ingredient. Both the crude root of Iris Florentina and the commercially prepared orris powder produced in these cases marked skin reactions and vasomotor disturbances. In several of these patients the cause of the symptoms had not been previously suspected, but in all instances a complete subsidence followed the discontinuance of the face powder.

(b) Of Animal Origin.—Forty-nine cases were found of sensitization to epidermal scales and hairs of animals or feathers of birds. The symptoms varied from vasomotor rhinitis to severe asthma when inhaling fine suspended particles of the special exciting substance in question. In all cases a definite reaction was elicited by the application of a keratin extract to a scratch of the skin. While the majority were aware of their sensitization, yet for a number of individuals the first recognition of the cause was obtained by the test. This was especially found in the case of children.

Of the various animals the horse was represented by forty-two cases, the dog by eight, and the cat by six. The sheep and cow gave in two cases respectively moderate but apparently definite reaction to keratin extracts from the natural hair, but it did not appear that the first case was affected by the dust from woolen blankets. In two cases definite vasomotor rhinitis was occasioned by the use of feather pillows, and keratin extracts from feathers of the chicken or duck gave well defined skin reactions. The symptoms disappeared on the discontinuance of the feather pillow.

A distinct difference appears to exist between the proteid of the serum and that of the epidermal structures in the animals studied. In the case of the horse, eleven patients gave marked reactions to the keratin extract but no reaction to the serum, either in pure powder or as antitoxin. That this difference is not due to greater strength of the keratin extract employed is shown both by the fact that the majority of patients gave equally marked reactions to both serum and keratin, and by the observation made in one individual of a pronounced cutaneous reaction to horse serum, without a trace of disturbance from the keratin. This patient had had but little to do with horses, but had thirty-eight years previously been bitten by a colt. It is conceivable that in this manner a sensitization to the serum alone may have developed.

While the majority of the dog and cat cases reacted to keratin, both clinically and by the tests, yet two individuals were discovered who reacted to dog serum in a marked degree without sensitization to dog hair. Two similar cases were found reacting to cat serum alone. These four individuals had no discomfort when in the neighborhood of dogs or cats respectively.

Of the two cases of feather sensitization, one showed no reaction to eggs.

These observations indicate the necessity of employing keratin extracts as well as sera when making diagnostic tests of sensitization. Furthermore, the probability is suggested that certain cases of horse fever or horse asthma may in reality receive antitoxin without anaphylactic shock if their sensitization is to the hair and not to the serum of the animal. Finally, the possibility is conversely suggested that the absence of symptoms from horses does not preclude the possibility of an anaphylactic shock from the administration of antitoxin.

II.—INGESTION ANAPHYLAXIS.

Twenty-eight cases were characterized by more or less well defined sensitization to various foods. This number does not represent the true relative frequency of such cases, since their study has been undertaken only during the past six months, and many of the earlier ones may have escaped recognition.

These cases represent all gradations ranging between sensitizations so slight as to have excited no subjective symptoms on the part of the patient to extreme susceptibility, in which contact of the smallest part of the proteid in question caused profound general disturbance. It was noted that the local sensations were referred to the throat rather than to the region of the stomach. Such symptoms were described variously as tickling or itching in the throat, or in more severe cases as swelling and smarting. The general constitutional phenomena were in some cases definite asthmatic symptoms, in others urticaria. Eczema was found associated in a number of instances, and psoriasis occurred once.

Animal foods were prepared for testing, as already described, and instances were found of sensitization to shell fish, crustacea, fish of various sorts, egg, milk and meat. If the skin reaction was marked, the patients were usually aware of their idiosyncrasy. This applies particularly to intolerance to fish and eggs.

Milk was found in one instance to give severe asthma in a child seven years of age. This patient had had more or less constant asthma since being weaned. Physical examination by Dr. J. L. Morse was negative. The skin tests showed reaction to milk, horse and cat keratin. On discontinuing the milk and keeping the cat away, the asthma ceased completely. At the end of two weeks the child was given cow's milk again, with a return of the asthmatic symptoms. This was allowed to continue for a week and the milk discontinued, the asthma simultaneously disappearing. A week later he was allowed to play with the cat, whereupon asthma reappeared. On keeping him away from the cat and giving milk at the beginning in drop doses and gradually increasing, it was possible at the end of six months to establish a tolerance for a glass of milk. The child has during this time been free from asthma.

Cases of sensitization to egg have been referred to children's specialists for immunization by progressive doses, with successful results.

A case of sensitization to raw animal sera was found in a boy twelve years of age. This individual reacted to dog, cat, hog, sheep and ox serum to a high degree. Horse serum and goat serum excited slight reaction. Guinea pig serum gave no reaction. Tests with cooked beef and lamb gave no reaction.

There was well defined sensitization to keratin of dog, cat, horse and lamb, but none to that of the ox. Commercial casein and egg gave no reaction. This case indicates the desirability of having the animal foods prepared for testing under the form in which they are usually taken into the system.

In the case of plant foods we have likewise to prepare the material in the raw, boiled or roasted state, as the case may be. A variety of proteid substances exists in plants, the greater part of which is coagulable by heat. While many proteid substances in aqueous solution require a temperature of 100° C. before being coagulated, others coagulate at 65° C. Proteoses withstand boiling, but may be precipitated by treatment with absolute alcohol. Eleven cases were found of sensitization to grains. In certain of these the patients had no subjective sensations. In four, however, it seemed probable that the asthmatic symptoms of which they complained were attributable to the eating of grains in the form of cereals and bread. Two of these cases were reported somewhat in detail.

Case 1.—G., aged forty years. Asthma since childhood, of unknown origin, more or less persistent throughout the year. No reactions were found to animal foods or to the pollen of grasses, and the patient had no hay fever. Raw and boiled proteids of wheat, barley, oats, corn and rice gave marked skin reactions. Proteids extracted from the inside of bread gave reactions much less pronounced than the raw or the boiled material. Extracts from well browned toast and some roasted grains of wheat, corn and rice gave no reaction whatever. Various breakfast foods were tested, shredded wheat, triscuit, corn flakes, force, Kellogg's wheat biscuit and puffed rice, giving no skin reaction. Grape nuts gave a pronounced reaction. The patient was negative for other plant foods with the exception of beans and peas. Bread and boiled cereals were omitted from his diet for a period of two weeks, during which time his asthma became much less, giving him at the close of this time practically no discomfort except on exertion. On eating again freely of bread, after two days the asthma returned in its original intensity, diminishing later when bread was omitted. When the roasted cereal foods above mentioned, which gave negative reactions, were introduced into the diet, there was no return of the asthmatic symptoms, and the patient is now in greatly improved general health.

Case 2.—S., aged thirty-five years. Asthma many years, more or less constant. Hay fever in spring, moderate. Marked reaction to wheat, raw and boiled, and to bread proteid. Marked reactions to raw and boiled proteids of oat, barley, rice and corn. Positive reactions were obtained also for cocoa and buckwheat. Squash, pea, almond, and Brazil nut gave pronounced reactions. There was no reaction to beet, turnip, cabbage, parsnip, potato, eggs, fish or meat. This case was relieved very much of his asthma on omitting bread and grains, and the other plant foods to which he was sensitized. A proteid extract of wheat was treated with pancreatin for three days. This modified extract caused marked skin reactions. Wheat, rice and corn heated to a temperature of 115° centigrade, gave no reaction, and their proteids on treatment with pancreatin for three days gave no reaction.

These two cases indicate that certain proteids present in the grains are heat stable up to 100° C., but at 115° C. their anaphylactogenic property is destroyed, and is not reintroduced by pancreatic digestion.

Four cases of sensitization to potato, both raw and boiled, were observed. In one of these only was there any systemic disturbance, this consisting in an urticarial eruption. Proteids from baked potatoes gave no skin reaction.

Cases were also encountered of sensitization to nuts, both walnuts, chestnuts and Brazil nuts, some reacting also to almonds, although this is derived from the rose family, and stands far removed in a botanic classification from the others. These cases did not react to the pollen of chestnut or the rose family respectively. A few cases were found of sensitization to strawberries, bananas, oranges and apples in the raw state. Cooking, however, of the strawberries and apples made it possible for the patients to eat the fruits without discomfort. The inference here to be drawn is that the sensitizing proteid was coagulated or destroyed on boiling. The cases of clinical sensitization to grains as a rule had no hay fever, and did not react to the pollen of grasses. It was, however, observed that cases of marked sensitization to grass pollen gave, without exception, a definite skin reaction to proteids, both boiled and raw, of grains. This observation suggests the probability that the pollen represents the specific plant proteid found throughout all its parts, as Metz and Gohlke have apparently shown, but that in the development of the ovule during the summer other proteids are formed with anaphylactogenic properties differing from those of the specific plant proteid, and that these seed and fruit proteids represent a high degree of differentiation among closely related species. This clinical observation receives confirmation from the works of Wells and Osborne. These observers have isolated from the seeds of closely allied plants substances resembling proteoses, which are distinguishable from each other. They reach the conclusion that resemblances and differences of these substances are dependent upon the chemical constitution of proteid molecule, rather than upon biologic differences. They, for instance, found that zein, the alcohol soluble protein of corn, did not cause the anaphylactic reaction in animals sensitized with gliadin, the alcohol soluble proteid of wheat, nor with hordein, the alcohol soluble protein of barley. At that time hordein and gliadin had not been tested against one another, but it had been observed that preparations of gliadin from either wheat or rye interacted against one another, as if they were one and the same protein. On testing hordein from barley against gliadin from either wheat or rye, they have since found that these two proteins of different origin also react with one another. Furthermore, they have found that while preparations of gliadin from wheat reacted anaphylactically with glutenin from the same seed, hordein from barley fails to cause reactions in guinea pigs sensitized with glutenin.

It has seemed to me possible that these relationships between the specific plant proteid found in the pollen, and those of the seeds and fruits may be represented graphically as follows: Taking for instance the Rosaceæ, we may let A represent the specific proteid of the family found in the pollen and throughout all the parts of the plant. B, C and D may be taken to denote the fruit proteids of the apple, strawberry and almond, elaborated by the activity of the respective plants during the summer's growth. The proteids present in the fruits will, therefore, be AB, AC and AD. An individual sensitized to the pollen of the rose will react positively to the pollen of all the members of that order, and will also react to extracts from the fruits or seeds of all. If, however, he is sensitized to proteid B of the apple, he will react neither to the pollen of any of the rose family, nor to proteid C of the strawberry, nor to proteid D of the almond.

This hypothesis has apparently received confirmation from my observations on the Rosaceæ and Gramineæ, as already mentioned.

III.—AUTOLYSIS OF BACTERIAL PROTEIDS.

While the influence of proteids from animals and from flowering plants in the causation of anaphylactic symptoms is sufficiently recognized, yet the part which possibly bacterial proteids may play in these conditions is not yet clear. Clinically we find a large number of cases in which the influence of bacteria seems probable. We may divide these conditions provisionally into acute recurrent and chronic forms. In the first instance we find that the individual goes for considerable periods of time with little or no vasomotor disturbance, and that with the advent of an acute infection of the nasal or bronchial mucous membrane, he exhibits symptoms of vasomotor rhinitis or of asthma. The mucous membranes in these conditions do not show reddening or pus formation which are characteristic of acute rhinitis and acute bronchitis, but present rather the typical picture of hay fever. After the cessation of the acute symptoms, the individual is free from asthma until the occurrence of another infection. The existence of chronic forms of bacterial infection have been recognized by clinicians as being represented on the one hand by chronic sinuitis and on the other by intestinal stasis, and we have probably all had cases where treatment or cure of these conditions has resulted in relieving the vasomotor symptoms. I believe that chronic lacunar tonsillitis with accumulation of detritus must be regarded also as a possible cause of proteid absorption.

In a recent paper I have called attention to the possible explanation of such types of vasomotor disturbance, referring them to the solution or autolysis of bacterial proteids to which the patient is sensitized. On this hypothesis both these acute and chronic forms of vasomotor rhinitis and asthma are dependent upon the occasional or constant anaphylactogenic action of autolyzed bacteria. Experiments which were then reported for the purpose of determining whether bacterial proteids in solution could elicit skin reaction, showed that out of fifty cases positive reactions were obtained from the staphylococcus pyogenes albus, aureus and citreus; also from a bacillus of the Friedlander type, and the micrococcus tetragenus. Since then further study has confirmed these observations, and reactions have been obtained in a number of cases from the bacillus of influenza, streptococcus pyogenes and viridans and pneumococcus, type I and II. It was also observed that after an attack of asthma the skin reaction could not be elicited, but returned again after a few weeks. Much still remains to be done before conclusions can be drawn as to the relation of these organisms to the vasomotor disturbance in question. If it should be confirmed that definite sensitizations to microorganisms exist on the part of these anaphylactic cases, our therapeutic work should receive a highly important assistance. We should, for instance, in the case of acute recurrent infections be guided in the choice and administration of vaccines. The cases of chronic proteid absorption would be more intelligently approached in that we should be led to examine the patient with especial care for the purpose of removing, if possible, the foci of infection, or at least of draining and cleansing the regions in which prolonged contact of bacterial proteids may occur.

ASSOCIATION OF FACTORS AND THE OCCURRENCE OF CLINICAL SYMPTOMS.

A review of the cases has shown that for the majority a variety of sensitizations is present. We find, for instance, often hay fever disturbances from the pollen of different orders, while here and there hay fever patients occur with sensitization also to bacterial proteids, certain classes of foods, or keratin desquamations.

In such cases our task is first to determine the factor which is responsible for the symptoms, and to place in proper perspective the relative influence of the other sensitizations to which the patient is subject. The line of inquiry is usually as follows: First, are the symptoms strictly seasonal? In this case we shall obviously be able to disregard all factors except the various pollens. If the symptoms are perennial, inquiry is next made as to their relation to the occurrence of colds and acute infections. If the answer is confirmative and the patient states he is entirely free from discomfort except when suffering from a cold or acute bronchitis, we may feel justified in disregarding the influence of both inhaled and ingested proteids, and devote our attention to an examination of his reactions to the various bacteria concerned in acute infections of the upper air passages.

If the symptoms are more or less constant throughout the year, the possible influence of domestic animals should be sought for, especially dogs and cats in the case of children. Nor must the possibility of orris root in toilet or sachet powder be forgotten. Inquiry is next made as to disturbance from different articles of foods, and here the tests should be first made for such substances as enter habitually into the diet: thus, in order of importance, we place eggs, milk, meat, grains, legumes and potatoes, since if the patient is not sensitized to any of these, it is unlikely that the more occasional articles of diet, such as shell fish, fish, root vegetables, greens and fruits are responsible.

If the above tests have resulted negatively, our attention should be directed to the possible existence of chronic absorption of bacterial proteids, and we may then proceed with the examination of the patient as to his sinuses, tonsils and gastro-intestinal tract.

THE MANAGEMENT OF PATIENTS SHOWING HYPERSUSCEPTIBILITY.

In symptoms arising from inhalation of sensitizing substances, if the exposure is likely to continue throughout the year, it is probably wiser to avoid the irritation than to seek to immunize the patient against it. We have at least as yet no record of successful immunization. A case which I reported last year of attempted immunization against horse asthma by the injection of horse serum and keratin extract showed a gain in tolerance to the presence of horses, but on the discontinuation of the treatment the immunity was gradually lost, and the patient has now regained her original sensitiveness. Since for the successful preservation of the required resistance continuation of proteid administration is necessary, and inasmuch as we have yet developed no successful method which the patients would be willing to carry out for an indefinite period, it would seem preferable for the patient to accept his disability, and make the best of it.

Immunization against hay fever by the injection of plant proteids has been described by other writers, and by myself in a previous paper, so that I shall not consider it here in detail. Before, however, we undertake the treatment, we should first decide whether the patient's discomfort is sufficient to justify us in starting him upon a course of procedure which may prove time-consuming and not wholly devoid of risk. The treatment is by no means to be compared to typhoid inoculation, but will require numerous visits, with careful tests of the degree of progress in order to determine the correct dose for each occasion. I cannot too strongly emphasize the importance of this matter of dosage. We are dealing with agents capable in moderate overdose of causing unpleasant shock, and in sufficient amount of leading promptly to a fatal result. The best advice that I can give one starting in this work is to test continually the patient's resistance, and to increase the doses gradually in conformity with the skin or conjunctival tests.

In reviewing the results thus far obtained in three years' treatment of hay fever by hypodermic injection, one must in the first place be guarded in accepting the statements of the patients themselves. As I have previously pointed out, seasons vary, and the physical condition of the patient may likewise differ from year to year. Patients are all too ready to attribute a greater freedom from hay fever symptoms to the treatment employed. It is only where the patient's statements are controlled by clinical tests that they may be accepted as possessing value. With this preliminary statement I will say that the majority of patients who received treatment in the past two summers expressed themselves as satisfied with the results accomplished. From those who felt themselves improved, however, a considerable number should be eliminated, as careful examination of their surroundings, the season and their physical condition might permit other explanations of the diminution in symptoms. The typical individual who presented himself for treatment two or three months before the expected season showed a progressive diminution in the intensity of the skin reactions, and a greater tolerance to the solution employed. In most instances, however, the patients did not escape entirely the hay fever symptoms when the season came around. The typical behavior was as follows: Hay fever appeared at the usual time or somewhat later, the latter possibly attributable to the season, and reached within a few days nearly its accustomed degree of intensity. This was, however, followed by a striking diminution in the symptoms, so that after the acme of discomfort had been reached, a very considerable degree of relief supervened, and most of these cases remained relatively or entirely free from trouble through the balance of the season. The impression was given in such cases that the amount of treatment which it had been possible to administer in the few weeks before the season was insufficient to effect a complete immunity, but that with the arrival of the season enough pollen was absorbed through the mucous membrane to push the patient, so to speak, over the line. The same results were apparently obtained when treatment was instituted at the beginning of the hay fever season, and in the majority of such instances it was possible to bring about a relief of hay fever symptoms several weeks before relief would ordinarily have been due.

I have received the impression, consequently, that we are not likely in the majority of cases, with the present methods, to do away completely with hay fever symptoms in the course of a single season, but that, nevertheless, sufficient relief may be obtained to make it worth while to continue this method.

Where, however, treatment has been carried on during one season with more or less success, it has been evident that a second course of treatment the following season is more distinctly efficacious.

The usual course of events here is as follows: At the close of the first hay fever season one finds that the patient has attained a relatively high degree of immunity which in the course of the winter progressively diminishes. Beginning again next season, several weeks before the expected date for hay fever, one injects a small dose, carefully determined in the usual way, and follows this by larger doses. In practically all such cases a rapid striking diminution in the intensity of the skin reaction was observed, much more speedy than was the case during the first season. Such cases when the hay fever season arrived were distinctly more benefited than during the previous year, more than half showing no or exceedingly trifling symptoms. The work is yet too young to enable us to say with certainty what the future will bring forth, but with our present information I should advise considerable caution in the statements to be made to the patients, and especially to explain to them that they may observe little or no benefit during the first season, but that the results obtained lead us to advise a following out of treatment for two years, with the reasonable expectation that the second season's treatment will be more helpful than the first.

Although feeding experiments in the past with extracts of the ragweed do not appear to have been sufficiently successful as to justify their general adoption, yet the work which internists have done with the treatment of egg sensitizations, has suggested the possibility that we may accomplish similar results by administering foods in a natural state derived from the order which produced the symptoms. Experiments were consequently undertaken during the past summer to determine whether the patients sensitized to ragweed pollen would present symptoms from the ingestion of proteid foods derived from the same family.

It was thought that if such internal administration was followed by the development of vasomotor symptoms, either of the skin or of the mucous membranes, an immunization might possibly be accomplished along the recognized lines of treating egg and other food sensitizations.

For this purpose sunflower and lettuce were taken, both being plants of the Compositæ, to which the ragweed also belongs. The pollen of both was found to excite marked skin reaction in cases of autumnal hay fever.

Extracts were made from the seeds of the Russian sunflower which gave positive results, but the intensity of the skin reaction was inferior to that excited by the pollen. The seeds were given to ten hay fever patients, with the direction that they should chew them thoroughly and swallow shortly before their regular meals, with the idea that a certain amount of the proteid might pass into the system directly without digestive alteration. In nine adult cases, however, no gastric discomfort was noted or any influence upon the hay fever symptoms. Oil-free meal from sunflower seeds was prepared which excited a moderate or marked skin reaction, depending upon the sensitization of the individual. This was administered internally in doses of one to two grams, without exciting symptoms of any kind whatever. One patient, a boy, said that his hay fever was made worse after eating the sunflower seeds.

In the case of lettuce, a strong extract was made from the seeds in a five per cent sodium chlorid solution, which gave a weak reaction. A strong solution of lettuce leaves in a ten per cent sodium chlorid solution (proteins shown to be present by preliminary test) gave a negative reaction.

The amount of proteid present in the lettuce leaf is evidently too small to excite any disturbance, and with one doubtful exception no patients had noted any influence upon their hay fever from eating it in abundance.

It would appear, consequently, probable that we have no foods of the Compositæ order, the ingestion of which is likely to influence the course of ragweed hay fever.

INGESTION.

In the course of this work it has become evident that many individuals exist who show definite anaphylactic skin reactions to various classes of foods, and the problem which here confronts us is to determine to what extent the clinical vasomotor symptoms are dependent upon such a hypersusceptibility. It has in some instances seemed indicated that certain individuals possess food sensitizations, either partially developed or measurably overcome through the acquisition of progressively increasing tolerance, in whom the actual vasomotor explosion is in reality dependent upon some other proteid cause. The following case illustrates this possibility:

D., male, aged forty years. Asthma severe, nearly constant for many years. Marked polypoid degeneration of middle turbinates with involvement of antra. Skin tests were positive for staphylococcus pyogenes albus and a diphtheroid bacillus; negative for pneumococcus, the influenza bacillus, micrococcus catarrhalis and staphylococcus pyogenes aureus. Positive reactions were obtained from proteid extracts of wheat, barley, corn, rice, pea, orange and cocoa. Negative or slight reactions were obtained from egg, milk, beef, mutton, fish, potato, carrot, beet, parsnip, squash and cabbage. Keratin extracts of domestic animals and birds were negative. The patient was put upon a diet containing only those foods to which he was negative, but without perceptible influence upon the asthma. No operation has as yet been done upon his sinuses, but in view of the above observations this seems the measure most reasonable to undertake.

Conditions of highly developed sensitization to foods have been studied by numerous observers, especially Schloss, Talbot and Chappell. It has been evident that children are much more prone to exhibit anaphylactic phenomena from foods than is the case in adults, a fact possibly due to the greater absorptive power of the gastric mucous membrane in children, or to the acquisition of partial immunity in adults.

If the proteid enters into the accepted articles of diet, experiments have shown that immunity can be obtained in the majority of cases by administering it in small and progressively increasing amount. The details of treatment may be found sufficiently described by the authors mentioned.

The management of cases in which the vasomotor symptoms either demonstrably or by exclusion are attributable to the absorption through the mucous membrane of bacterial proteids is to be undertaken along lines already indicated. Acute recurrent infections may perhaps be checked by the administration of vaccines. The sensitized individual presents here an advantage, in that it may be found possible to select for him with accuracy the special organism required.

Foci of chronic inflammtaion or sepsis should be drained or removed. In three of my cases of long standing asthma, the excision of septic tonsils was followed by entire relief.

Cases of intestinal stasis should be treated by the internist or the orthopedic surgeon, as circumstances may require. Two cases of asthma with ptosis of the viscera were relieved by the application of a proper support.

CONCLUSIONS.

In a large proportion of vasomotor diseases of the upper air passages the disturbances are dependent upon the entrance of a foreign proteid into the system. The method of entrance may be through the contact of the proteid in question with mucous membranes of the respiratory or of the gastrointestinal tract by inhalation or ingestion, respectively. Foreign proteids may perhaps also develop in or upon these mucous membranes through autolysis of pathogenic or saprophytic bacteria. The application of the skin test to these conditions is of diagnostic value when employed with a recognition of the phylogenetic relationships of animals and plants as determined by studies in serobiology.

Proteid material for testing should be prepared both from the keratin and sera of domestic animals, from the pollen of the chief causes of hay fever, and from the various articles of food, which enter commonly into the diet. Bacterial proteids derived from the various invaders of the respiratory tract should be available either in solution or in the soluble form.

When the skin reactions to the various classes of pollen proteids have been determined, the management of cases will depend largely upon the relative preponderance of the local reactions in relation to the clinical history. If the cause is found to be seasonal, as in hay fever, immunizing treatment by injection of pollen extracts is likely to prove of service. The sensitization returns during the following winter, and treatment must probably be repeated annually. If the cause is perennial and is due to the inhalation of foreign proteids, it is wiser to avoid the cause rather than to seek to effect a cure by immunization. If the disturbing proteid enters into the ordinary articles of diet, a tolerance may be gradually established by feeding the substance in progressively increasing doses. Disturbances of bacterial origin have not yet been sufficiently studied to enable the formulation of a definite plan of treatment, but the results of these investigations confirm our present methods of treatment, and emphasize the importance of draining regions which can retain the products of bacterial activity. Septic foci should be removed. Vaccine therapy is likely in such anaphylactic cases to be more accurately guided than in the ordinary individual.

The results already accomplished in the case of this clinical investigation have led me to the conclusion that we possess in the intelligent application of the skin test a very definite aid in the diagnosis and consequent management of our cases of vasomotor disturbances of the upper air passages. We are as yet only at the entrance of this field, and its exploration must be carried out by the cooperation of laboratory workers in chemistry and serobiology.

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XXVIII.

ANATOMIC RELATIONS OF THE CAVERNOUS SINUS TO OTHER STRUCTURES, WITH CONSIDERATION OF VARIOUS PATHOLOGIC PROCESSES BY WHICH IT MAY BECOME INVOLVED.*

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The object of this paper is to present as clear a picture as possible of the anatomic relations of the cavernous sinus to adjacent structures, and to index in one article, as far as possible, the scattered data of pathologic affections available in the literature of the past fifteen years, together with the important addition of unpublished information obtained from question-letters sent to prominent general surgeons throughout the country. The idea of instituting a search of this kind grew out of the fact that in reading a paper on "Cavernous Sinus Thrombosis and the Future," before the Chicago Ophthalmological Society, November, 1914, the discussion seemed to indicate: First, that while most of us were pretty well informed so far as one or two diseases of the sinus was concerned, our information stopped quite abruptlly at that point; second, that deductions from individual experience was extremely limited; and, third, that possibly statistics with reference, at least to mortality in septic thrombosis of the sinus, might not be accurate. Because of the speculation and the rather chaotic condition of literature, the writer determined to investigate the subject more fully, and to present such matter as could be gathered, even though the results obtained might not prove in the end worth the labor involved. With this preliminary statement, therefore, such material as has been gathered is herein presented.

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ANATOMY.

The cavernous sinuses, two in number, one on each side of the sella turcica,* are a part of the general venous system at the base of the skull. Like the other large venous channels, they are situated between two layers of the dura (splitting of the dura), are lined by endothelium continuous with that which lines the veins generally, and serve to drain the blood anteriorly from about the orbits, and anterosuperiorly from the brain itself. These sinuses carry the bulk of this venous flow backwards through the petrosals to the internal jugular veins, the great outlet for the cerebral circulation. Their anatomic importance depends upon their location in the middle cranial fossa, just lateral to the body of the sphenoid bone, extending from the sphenoidal fissure in front to the apex of the petrous portion of the temporal bone behind, containing venous blood, and being traversed by certain other (transient) structures and being connected internally with each other by communicating small intercavernous sinuses which at the same time surround the hypophysis cerebri.

The cavernous sinuses are so named because they present the picture of a reticulated structure† traversed by numerous interlacing filaments which divide the sinus cavity into irregular compartments. The sinuses are larger behind than in front and measure from two to two and one-fourth centimeters in length, and have a diameter of about one centimeter. A cross section shows these sinuses to be of such shapes as quadrilateral, shallow S outline, or distinctly tooth shaped. As expressed by Piersol[1]: "Its external diameter does not represent the actual capacity of the lumen, since this is greatly reduced in size: (a) by being traversed by numerous trabeculæ from which fringe-like prolongations hang freely in the blood current, a section of the sinus having very much the appearance of a section of the corpus cavernosum of the penis, whence the name bestowed upon it by Winslow; and (b) by the fact that the internal carotid artery and abducens nerve traverse it, while other cranial nerves are imbedded in its outer wall."

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*Sella turcica (Turkish saddle)—the depression within the three clinoid processes of the sphenoid bone lodging the pituitary body.

†As a clear mental picture of the sinus is important for proper clinical application in disease, it is worth repeating with Halstead that anyone "who has dissected the cavernous sinus will realize that it is not a single cavity, but it is a sinus made up of a number of cavities—from one to fourteen separate cavities, connected by a very small opening, so that you may open and scrape out one of these cavities and still leave others that are infected and continue to spread infection." J. C. Beck has also emphasized the fact that in attempted drainage of the sinus we must take this partition idea into consideration, for in instituting any drainage which might be successful the sinus must be drained as thoroughly as any other thrombotic vessel and that it would be extremely doubtful whether a small slit or puncture in one of these partitions would really accomplish much good.

Anteriorly each cavernous sinus receives as tributaries the ophthalmic vein (through the sphenoidal fissure), sphenoparietal sinus, lodged in the dura on the under surface of the lesser wing of the sphenoid, and a smaller cerebral vein (vena cerebri media and sometimes vena cerebri inferior). Anteriorly the sinus is in direct communication with the angular vein at the inner angle of the orbit through the superior ophthalmic vein.

Posteriorly the cavernous sinuses terminate by opening into the superior and inferior petrosal sinuses at the junction of the petrous and sphenoid bones (foramen petrosphenoidale). The cavernous sinus communicates with the lateral sinus by means of the superior petrosal sinus; with the internal jugular vein through the inferior petrosal sinus, and through a plexus of veins on the internal carotid artery (Gray[2]); and with the pterygoid plexus through the foramen ovale.

Internally the two sinuses communicate with each other by means of the intercavernous sinuses (anterior and posterior intercavernous sinuses, with at times a third or inferior intercavernous sinus). The internal surface has anywhere from three to seven openings by which it communicates with its fellow on the opposite side and also with the hypophysis cerebri. On the inner wall within each sinus is found the internal carotid artery, accompanied by filaments of the carotid plexus and the abducens nerve. The communication also between the two anterior ends of the inferior petrosal sinuses at the posterior extremity of the cavernous by the plexus basillaris (or so called transverse sinus) is another anastomotic connection which tends to equalize and distribute blood pressure for the larger cavernous.

The external or outer wall of the sinus is occupied by the oculomotor, trochlear, ophthalmic and superior maxillary divisions of the trigeminal nerve. All such structures, however, are separated from the blood flowing through the sinus by the endothelial lining membrane continuous with the inner coat of the veins previously mentioned.

Summing up it may be said that the transitional structures of the cavernous sinus number seven in all—i. e., internal carotid artery, carotid plexus, abducens, oculomotor, trochlear, ophthalmic, and superior maxillary nerves.

The upper three-fourths of the external surface of the sinus is in relation to the internal part of the temporosphenoidal lobe, while the lower one-fourth is in relation to the inferior maxillary nerve (mandibular branch).

The superior border of the cavernous sinus is in relation in the posterior four-fifths with the dura covering the cavernous portion of the oculomotor nerve, and in the anterior one-fifth with the lacrimal nerve. The inferior border is said to be "formed posteriorly by endothelial lining membrane being reflected on the intracranial portion of the nervus maxillaris." (Hoeve.[3])

Relation of Structures in the Cavernous Sinus.—As outlined by Hoeve,[3] in the posterior four-fifths of the sinus, from above downward, the oculomotor, trochlear and ophthalmic nerves rest against the internal surface of the outer wall. The carotid artery, surrounded by the carotid plexus and the nervus abducens, rests against the internal wall, and the plexus cavernosus lies internal to the carotid artery.

In the anterior one-fifth of the sinus the oculomotor nerve and the ophthalmic (first division of the fifth) divide into their branches within the sinus at the junction of the posterior four-fifths with the anterior one-fifth, and this breaking up into branches is the cause of the difference in relations in the sinus and the fissura orbitalis superior.*

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*Dissection (from Manual of Dissection and Practical Anatomy of the Head and Neck, Hubertus J. H. Hoeve, edition 1910, pages 347-349):

(1) Incise dura carefully along the superior border of sinus cavernosus, but do not cut the ligamentum petrosphenoidale at the posterior extremity of the sinus, which bridges over the interval between the apex of the petrosa and the base of the processus clinoideus posterior, and which is covered only by dura in that region.

(2) Reflect the flap of dura outward and downward and expose the interior of the sinus, and by breaking down carefully some of

Variations of the Cavernous Sinus.—According to Quain (as quoted by Mosher): "The cavernous sinus is represented in a child by a venous plexus in the dura mater, which by enlargement and fusion of its channels is converted into the characteristic sinus of the adult. In old age the trabeculæ diminish and the cavity becomes simpler." This is also confirmed by von Lauger,[111] based on corrosion and other preparations from infantile subjects. Instances have been noted where one cavernous sinus is quite rudimentary, leaving practically but one functionating vessel.

Relations of Cavernous Sinus and Transient Structures to Sphenoidal Sinus.—The cavernous sinus in a considerable part of its course overlaps slightly the roof of the sphenoidal cavity and is separated from this air cavity by the thinnest of bony partitions. According to Gibson,[4] from an examination of eighty-five sphenoidal sinuses, the roof of this sinus varies from 0.25 millimeter to two millimeters, averaging approximately one millimeter in thickness. Bony dehiscences have been observed by J. C. Beck and others between the sphenoidal sinus and carotid canal, in which the internal carotid artery might easily have been injured by too forcible instrumentation in the sphenoidal cavity. In most specimens the casing of the bony carotid canal is plainly seen bulging into the cavity of the sphenoid sinus near the posterior external angle. For the most part, therefore, it may be said that the cavernous sinus occupies a fairly constant position above and lateral to the body of the sphenoid bone (junction of roof and external wall of the sphenoidal cavity), and given a large sphenoid cavity, not only will the cavernous sinus be almost in contact, but also with any kind of a posterior prolongation in the body of the bone the basillar plexus may be in relation to the sphenoidal sinus as well. Indeed, diverticula are so frequently present in many sphenoids examined that through them quite a part of the circumference of the cavernous sinus region may be affected, bringing at times most all of the transient nerve trunks of the

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the trabeculæ find the nervus abducens and the sinus petrosus inferior as they pass through the foramen petrosphenoidale inferior to the ligamentum petrosphenoidale.

(3) Find that all the structures passing through the sinus are covered by the lining epithelium of the sinus, and so not in contact with the venous blood.

cavernous sinus or even the Vidian canal (Sluder[5]) and Gasserian ganglion (Mosher) in relation to the sphenoidal cavity.

CONSIDERATION OF INDIVIDUAL STRUCTURES.

Intercavernous Sinuses (Circular Sinus).—The circular sinus (anterior, posterior and frequently inferior intercavernous sinuses), which connects the two cavernous sinuses, is found in the sella turcica, surrounding the hypophysis cerebri and within the attached border of the diaphragma sellæ. It is formed most often by the three sinuses mentioned: the anterior intercavernous sinus, usually the largest, passing in front of the pituitary body; the posterior intercavernous sinus behind, and the inferior intercavernous sinus below this body. These sinuses receive branches from the pituitary body and from the dura mater. The intercavernous sinuses, together with that portion of the cavernous sinus between their terminations, form what is usually termed the circular sinus, although in reality it represents only a part of the extensive venous system around the body of the sphenoid bone.*

Basillar Sinus (Plexus basillaris—Transverse Sinus).—The basillar plexus consists of several interlacing veins resting on the dorsum sellæ (mostly basillar process of the occipital bone), connecting the two anterior ends of the inferior petrosals, close to the posterior extremity of the cavernous sinus. They lie between the posterior clinoid processes and the anterior margin of the foramen magnum, and communicate also with the anterior spinal veins. The basillar plexus can be detected by its bluish color when the dura is carefully removed in this region. Like the cavernous sinus, the basillar sinuses[111] are persistent channels of what was originally, in the fetus, an extensive venous plexus.

Superior Petrosal Sinus.—The superior petrosal sinus lies along the superior border of the petrous portion of the temporal bone (superior petrosal sulcus), in the anterior part of the attached margin of the tentorium cerebelli. It is small and narrow and serves to connect the posterior extremity of each corresponding cavernous and the bend or knee of the lateral sinus. It is a little above the petrosphenoidal foramen and trigeminal nerve. It receives some cerebellar and inferior cerebral veins, and veins from the tympanic cavity. According to Page,[23] the superior petrosal is frequently found to empty itself into the lateral sinus, not by a single opening, but through a series of openings three or four millimeters apart, extending along the sinus wall at a distance of two or more centimeters.

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*The two middle cranial fossae are connected with each other by the sella turcica, which contains the hypophysis cerebri and is bridged over by the diaphragma sellæ. The little oval opening in its middle is the incisura diaphragmæ sellæ, through which passes the infundibulum. The entire hypophysis is surrounded by venous sinuses (intercavernous sinuses) located in the walls of the hypophyseal fossa.

Inferior Petrosal Sinus.—The inferior petrosal sinus, larger but not as long as the superior, forms the shortest connections between the cavernous sinus and internal jugular vein. It is situated in a groove (inferior petrosal sulcus) formed by the petrooccipital suture. The inferior petrosal passes anteriorly through the foramen petrosphenoidale along with the abducens nerve, and behind through the anterior compartment of the jugular foramen, ending in the commencement of the jugular vein. The anterior ends of the inferior petrosals are connected with each other by the interlacing vessels of the plexus basillaris. The inferior petrosals receive as tributaries some inferior cerebellar veins, veins from the internal ear, pons and medulla.

Sphenoparietal Sinus.—The sphenoparietal sinus and ophthalmic veins may be regarded as the anterior beginning of each cavernous sinus. The sphenoparietal sinus is lodged in the dura below the inferior surface of the lesser wing of the sphenoid. It is located above the oculomotor nerve, and has as tributaries small veins from the diploe and the middle meningeal.

Ophthalmic Veins.—(a) The ophthalmic veins, superior and inferior, according to Gerrish,[6] have no valves, and the blood, under certain conditions, may flow from behind forward into the angular vein or its branches, and thus obviate pressure in the veins of the retina when the cavernous sinus is obstructed. The superior ophthalmic, beginning as the nasofrontal, communicates with the angular at the inner angle of the orbit. It also anastomoses with the inferior ophthalmic. Forming a short single trunk, it passes through the inner extremity of the sphenoidal fissure to the cavernous sinus. The swollen end of the superior ophthalmic, just before it enters the cavernous, has been dubbed sinus ophthalmicus.

(b) The inferior ophthalmic vein receives veins from the region of the floor of the orbit, and either passes through the sphenoidal fissure to join the pterygoid plexus of veins, or more directly backward through the sphenoidal fissure to the cavernous, either separately or by forming a common trunk with the superior ophthalmic.

Emissary Veins.—The emissary veins are vessels which pass through foramina in the skull wall to connect the cranial sinuses with the veins of the diploe and veins external to the brain (as in and about the scalp). These are important, in that they may serve as possible channels along which infection may be carried occasionally from the surface to the interior of the cranium. They are not always constant as to presence or location. The cavernous sinus, through emissary veins, is connected with the pterygoid plexus through the foramen ovale and Vesalius, as well as through the inferior ophthalmic vein; with the pharyngeal plexus through the foramen lacerum medius, and with the internal jugular vein by the carotid plexus through the carotid canal (Gerrish[6]).

Carotid Artery.—The second or petrous portion of the carotid is within the carotid canal, while the third or cavernous portion is that part of the vessel lying within the cavernous sinus along the side of the sella turcica. The artery first enters the bony carotid canal in the petrous portion of the temporal bone, and follows its windings, first upward a short distance, curving forward and inward, and again ascends to enter the cranial cavity in its cavernous portion. In the cavernous portion the artery follows the carotid groove or sulcus along the lateral body of the sphenoid extending from the posterior clinoid process behind to the anterior clinoid process in front, at which latter point it again curves upward and slightly backward to perforate the dura forming the roof of the sinus. After piercing the dura it passes between the optic and oculomotor nerves and runs to the internal extremity of the fissura lateralis cerebri, where it finally divides into terminal branches. Just at the point where the artery turns backward and internal to the anterior clinoid process, it may be easily mistaken for the ophthalmic artery (Hoeve[3]). The abducens nerve passes along with the carotid in the cavernous portion and external to it. Sympathetic filaments from the carotid plexus also pass with the carotid artery. In the cavernous portion the distribution of the carotid branches are as follows: Few small arteries to the hypophysis cerebri, Gasserian ganglion, arteriæ receptaculi, anterior meningeal and ophthalmic. According to Piersol,[1] where the internal carotid enters the cavernous sinus at the internal orifice of the carotid canal, the sinus projects downward around the artery in a funnel-shaped manner, and from it there arises a close network of veins, the carotid plexus or carotid sinus, which completely invests the artery throughout its course through the carotid canal, at the lower opening of which it is continued into one or two veins which open into the internal jugular.

Carotid Plexus.—The carotid plexus of nerves, situated on the outer side of the internal carotid artery, communicates with the Gasserian ganglion, sphenopalatine ganglion, sixth nerve, Jacobson's nerve, glossopharyngeal, and distributes filaments to the wall of the carotid artery and to the dura. Filaments of the carotid plexus may occasionally form a small ganglion (carotid ganglion) on the under surface of the artery. The relationship of these portions of the sympathetic nervous system to other adjacent structures of this region has been well worked out by Haskins.[7]

Cavernous Plexus.—The cavernous plexus of nerves is located below and internal to the carotid artery within the cavernous sinus. It communicates with the third, fourth, ophthalmic division of the fifth, sixth, ophthalmic ganglion, besides distributing filaments to the wall of the carotid artery. It is formed chiefly by the internal division of the ascending branch of the superior cervical ganglion. Its physiology belongs to the physiology of the sympathetic nervous system.

PATHOLOGIC AFFECTIONS OF THE CAVERNOUS SINUS.

The following data relating to diseases of the cavernous sinus have been obtained from question-letters sent to members of the American Surgical and Western Surgical Associations, and from the scattered monographs of affections available to me in the literature of the past fifteen years. While the information is not especially voluminous, it may serve as a basis to build upon in attempting the study of the cavernous sinus as a distinct and, one might say, separate organ of the body. As illustrating the difficulty and disappointment in gathering material, I would quote an extract from a letter from Dr. Louis B. Wilson, director of laboratories at the Mayo Clinic, Rochester, Minnesota, who reports that "No specimen, either from operation or autopsy, showing pathologic affections of the cavernous sinus has come into the laboratory since I have been connected with it, now ten years."

Out of two hundred and eighty-four letters mailed to members of the American Surgical and Western Surgical Associations, one hundred and twenty of the men responded, but of the number responding only forty-four were able to cite cases either directly or indirectly concerning the sinus. The classification of affections from the replies received from these letters is as follows: (1) Septic thrombosis, (2) gun shot wounds, (3) involvement from fracture of the skull, (4) arteriovenous aneurism, (5) injury during miscellaneous operations, (6) involvement by malignant growths, (7) congenital affections. To this from the literature gone over must be added (8) marasmic or socalled aseptic thrombosis and for the present (9) intermittent exophthalmos, both of which last are rare conditions. The foregoing admits, therefore, of a classification of nine separate and distinct parts, all worthy of attention, and is so far as I know practically the first attempt of its kind to systematize, and it is only a beginning.

(1) SEPTIC THROMBOSIS.

This subject has been pretty well thrashed out of late, so far as the ordinary avenues of infection are concerned, and the few case reports yearly do not add a great deal to our present knowledge. The principal question at issue concerns itself with early reasonably positive diagnosis, and whether successful treatment by direct operation with the many difficulties which attend, is feasible or not. So far, attempts in this direction have rather dismally failed.

From the question-letters sent out, which means mostly unpublished information, twenty-nine cases were reported to me of septic thrombosis, with recovery in three cases (Scholz, Kanavel, Halstead—see Table 1), or 9.6 per cent. Of these cases of recovery, one from the ear recovered through energetic and thorough aural surgical procedures alone—i. e., radical mastoid and jugular ligation operation; another, neck infection, recovered through local neck drainage, and the third, complicating an ethmoid operation with syphilis, got well on expectant, purely medical treatment. A direct operation was not attempted by any one of the twenty-one general surgeons upon whose data part of this report is based. (Table 1.)

In forty-four cases of septic thrombosis selected from the index of literature here appended (Johnson,[29] Adair-Dighton,[38] Bircher[107]) three recovered, or a percentage roughly of seven. Jackson[54] also gives a percentage of recoveries of about seven per cent, while Dwight-Germain[104] place their recoveries in a total of cases looked up as 7⅓ per cent, which seems to be not far out of the way and settles, I think, from the statistical point, that a patient does have some chance to recover, which some of us have scarcely thought possible.

To continue the subject farther. While it is not my purpose to present a too lengthy discussion of this topic, this article would be incomplete without some general consideration of the condition and some statement as to just how thinking men are beginning to view the matter of septic thrombosis about the body of the sphenoid bone. As has been alluded to elsewhere, physicians have felt and are feeling the need of more definite moral support, both in the matter of fairly early reasonable positive diagnosis, as well as from the standpoint of any line of treatment which might be contemplated when confronted with this disease. It may be mentioned also at this time that the problem of septic thrombosis is a borderland one, belonging to various fields, with, however, the striking features chiefly ocular. In consultation, therefore, we should, first of all, include the ophthalmologist for his eye findings, even though the actual charge of the case be placed in the hands of the general surgeon doing a good deal of head or pituitary body work, or of the rhinootologist specializing on the anatomy of the sphenoid region. This certainly, with some acquaintance with literature, would probably provide the best basis to work upon for intelligent future investigation.

Rarity of Cases.—The study of cavernous sinus thrombosis has been limited in the past (1) because the cases are rare and the average physician, even in a lifetime, will scarcely see more than two or three sufferers with the disease; (2) being surgically less approachable perhaps than almost any other structure in the body, few men really consider the sinus available for surgical interference, especially when the patient may seem already in a septic moribund condition and the cavernous sinus itself only one of a number involved.

Etiology.—For practical working purposes we may consider infection of this sinus as arising from two general directions: 1, posteriorly from about the mastoid or ear region from a thrombosed lateral sinus, the infection spreading directly forward against the blood current along one of the petrosals; 2, anteroinferiorly and a little less common by the infection from a carbuncle or wound of the face, jaws or nose, being carried backward, upwards and internally to the interior of the cranium along the drainage system of the ophthalmic vein or by anastomozing veins directly upward from about the throat. , The origin of infection at a remote region of the head or neck is one of the most important and tangible clues in diagnosis.*

Symptoms for Reasonably Early Positive Diagnosis.—The very first question that confronts us is the necessity of making a reasonably positive diagnosis fairly early in the disease, so as to make it worth while. As the findings of the ophthalmologist are always awaited with a good deal of expectancy by the other consultants, any opinion of value must needs show considerable acquaintance with the literature, a watchful lookout for just such a possibility under certain conditions, and, most important of all, getting every bit of information which is possible to obtain in any way about the patient before rendering a decision as to what is clearly taking place. Since the later classical signs with which many of us are familiar are among nature's last efforts, not until we have the most careful analysis of earlier symptoms, combined with almost intuitive judgment, can we expect to improve in diagnosis—and improve we must, if we would expect hopes of any future successful line of treatment. While medical men are naturally averse to taking the positive side in medicine, the fact that so many physicians stand by without feeling sure of their ground until autopsy, even after unmistakable symptoms are presented, often a number of days before death, makes the positive position for the skilled diagnostician all the more necessary just at present. One thing perhaps that has caused a good deal of confusion has been variations in the early onset of the disease, according to the origin of the infection, and also variations due to the intensity of infection.

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*The literature of septic thrombosis is well covered (successive articles) in numbers 104, 72, 68, 54, 37 of the index and the present paper.

So many factors enter into the causation of earlier symptoms that with the exception of beginning exophthalmos and chemosis of the lids of one eye, any one or often several of other signs, such as strabismus (internal or external) from paralysis of one of the nerves in the sinus wall, mild bilateral fundus congestion or marked papillary edema, meningeal symptoms, etc., may at first be absent. In a general analysis of cases the order of appearance of symptoms is about as follows: Deep seated pain about the eyes, septic temperature, varying widely with respect to degree, slight orbital congestion, lacrimation, conjunctival edema, a little lid swelling, slight ptosis, and beginning exophthalmos. The foregoing, associated for the first time with a more distant infective focus about the head or neck, at once suggests that all is not well, and that thrombosis of the cavernous sinus is developing. Difficulty in swallowing, neck tenderness, and some swelling on the infected side may also be present in certain cases and absent in others. Increasing lid edema and exophthalmos very quickly presents a not-to-be-forgotten picture. After thirty-six or forty-eight hours, beginning involvement of the opposite eye by lid chemosis and exophthalmos is noted, which is easily seen to come from behind and not to have jumped across the bridge of the nose, and is so characteristic as to admit of little argument in diagnosis. Pupillary changes, marked papillary edema, meningeal or brain abscess symptoms, metastasis in a distinct joint, septicemia and death is the final culmination.

In making a diagnosis of a given case, even without previous experience, one may formulate this rule: When we have increasing exophthalmos and lid edema on the same side as a somewhat distinct infecting septic focus, any edema of the face and neck, septic temperature, and the slightest evidence of beginning lid edema or exophthalmos of the opposite eye, we are safe in saying that the later positive symptoms of the cavernous sinus thrombosis are present beyond a doubt. A fairly early diagnosis in cases where the infection originates posteriorly about the ear or back of the neck is comparatively easy, and should present no difficulty. Cases arising anteriorly about the face, nose or jaws, however, present at times considerable difficulty, especially if the focus of infection is in or very close or the orbit. In some instances where the trouble started anteriorly even, yet owing to our lack of individual clinical experience, it is practically impossible to be certain of the diagnosis until the second or opposite eye begins to show some slight involvement by lid chemosis and exophthalmos. It is in these instances, however, where intuitive judgment points out what might very well happen under suspicious circumstances, as much as any reliance upon ophthalmoscopic evidences of bilateral venous stasis within the eyeball. While the condition is to be differentiated from septic infection of the frontal sinus, beginning erysipelas, orbital cellulitis and a few sphenoidal conditions, in reality these should be ruled out without much difficulty.

Prognosis.—The prognosis is bad, and the mortality for all working purposes, under present conditions, should be considered much higher than our statistics of seven per cent would indicate. Indeed, for all practical purposes in an individual case we have little reason to hope for a recovery. Medical science has not as yet attempted a systematic campaign to solve the problem of earlier diagnosis or practical treatment. In literature of recent years and from private sources (Table No. 1, and References Nos. 29, 38, 107) I have obtained six instances where a diagnosis of septic thrombosis of this sinus has been made and nature was able to overcome the infection and the patient lived. In these instances attention was given solely to the original infecting focus, and not to the cavernous sinus itself. Of seven further attempted operations of any kind to drain the sinus by direct surgical procedure (Bircher,[107] Voss,[108] H. Knapp,[105] Dwight-Germain,[104] C. A. Ballance, Ballance-Hobhouse,[37] and Mosher,[25] all have failed to accomplish the purpose, and two at least we know failed even to get to and open the sinus toward which the operation was directed.

In correspondence last year with five each of our prominent general surgeons, pituitary body workers, otorhinologists, anatomists and neurologists, eleven reported no personal experience with the disease and would make no comments; five believed the sinus might be amenable to treatment, and nine were most skeptical and expressed themselves as against any operative procedure or real hopes for the future along this line. As possible operative routes to attack the sinus or methods of treating same the following were mentioned as still to be perfected: Mosher's proposed anterior route through the orbit, Hartley-Krause[112] Gasserian ganglion route through the side of the skull, Luc[81]-Langworthy[68] routes upward through one side of the nose and sphenoidal cavity, and lastly, in certain otitic cases, energetic treatment of the original focus alone from behind—namely, by opening the lateral sinus and jugular bulb thoroughly and resecting the internal jugular vein.

Recognition of Factors Necessary for Real Progress.—From the foregoing it must frankly be stated that no substantial support can be deduced from past experience. What little there is in literature is all against expected saving of life by any special operative endeavor on our part beyond thorough operative exposure of the site of original infection. In reality, reports would seem to indicate that such cases "represent organisms which are ruined and in which all science loses its right to interfere." In the face of this, however, the few men who have studied the question most thoroughly and attempted something in the way of relief seem loath to accept such an unalterable dictum, but cling to the hope, even though not very well founded, that little will be lost by following a more active and logical operative plan until future systematic demonstration of failure—not in one or two scattered instances, but from dozens of attempts made all over the world and under all kinds of conditions—has settled the question more finally. It certainly is time, if we are to progress in our handling of so baffling a disease, that we must tenaciously adhere to three cardinal points connected with it: (1) Standing flat footed upon a reasonably positive diagnosis as early in the disease as possible; (2) viewing the sinus as we would any other large vessel which has become thrombotic; (3) following routinely that operative path which would seem to the surgeon in charge as best fitted for drainage. This last will naturally vary, operators preferring methods as selected and reported by themselves.

Limitations of Deductions From Purely Cadaver Work.—Last year the writer endeavored by systematic work on the cadaver to once more approach the matter of practical anatomic access in as unbiased a manner, and choose, if possible, the best, if one be here allowed such a term, of the three routes offered so far—i. e., (a) Hartley-Krause Gasserian ganglion route through an osteoplastic resection of the side of the skull; (b) Mosher's proposed anterior orbital exenteration and chiseling through the orbital plate of the great wing of the sphenoid; and (c) the nasosphenoidal route as outlined by Langworthy,[68] but which the pituitary body workers alone have indirectly demonstrated as containing any real practicability on a living subject. From the foregoing it may be stated that the surgeon, having specially prepared himself for the operation, is able to tap the cavernous sinus from any one of the three above directions, but not by any means easily. Under favorable conditions my own selection, in addition to thorough opening of the original focus of infection, would certainly be as follows, if given an actual case:

Writer's Proposed Operation.—Light ether vapor anesthesia. Plugging of posterior nares on one side and free injection of adrenalin chlorid solution about the operative region. Quick removal of obstructing ethmoid labyrinth, middle turbinate and anterior sphenoidal wall by the use of ethmoid curette, turbinate forceps, sphenoidal curette, punch, and long narrow-handle gouge and hammer. Once in the sphenoid cavity the author's straight and angular blunt curettes with overhanging edge[68] can be pushed through the roof of the sphenoidal sinus, close to the junction of its roof and external wall. The blunt ends of these curettes will push the carotid artery aside without damage, and by rotating the spoon in a forward direction away from the carotid artery, the overhanging edge of the curette catches bone and a hole of some size can be made leading directly into the sinus. This can be further enlarged by use of curettes, assisted, as mentioned, by a long narrow chisel and hammer. The chief danger of the operation here, I would say, is not quite so much the thick walled large carotid artery, but rather some unexpected small branches given off frequently in this region which when accidentally torn by instrumentation might prove troublesome or opening only a partially thrombosed sinus—much of this, however, is at present pure speculation.*

Cavernous Sinus Thrombosis and the Future.—In concluding the subject of septic thrombosis, I would repeat that the future alone must determine the real practicability of attempted earlier diagnosis and direct operation. At the present time, and looking fearlessly into the future, it would seem wise, until more evidence to the contrary is produced, to look upon these cases with increasing hopefulness and as offering at least occasionally surgical opportunities which should not be ignored.

(2) GUN SHOT AND OTHER PENETRATING WOUNDS.

Eight cases of gun shot wounds were reported by letter in which the sinus had been penetrated and otherwise badly injured. Six cases died, and two (Raymond, Halstead) recovered. (Table 2.)

(3) INVOLVEMENT BY SKULL FRACTURE.

Fracture at the base of the skull, with moderate laceration or complete rupture of the sinus, was reported in six cases. Two cases (Ochsner, Bevan) recovered, one by tamponing the sinus and the other by expectant treatment with, however, the final loss of the eye itself. (Table 3.)

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*Luc, of Paris (1905), proceeded by making a large opening through the anterior wall of the maxillary sinus on the same side as the cavernous sinus which he wished to open, and another smaller one into the nose, to locate the anterior wall of the sphenoid. Using a straight chisel, however, he was unable to penetrate the lateral wall of the sphenoidal cavity on that side, and so came to the conclusion that the cavernous sinus should be approached through the maxillary antrum on the opposite side of the head. This gave him the desired angle (oblique), and he was able on the cadaver to open the side of the sella turcica and penetrate the sinus without injuring the important structures. Brunert also seems to have had a clear idea of some time opening the cavernous sinus through its relationship with the accessory sinuses of the face. My own interest in this subject dates from seeing my first case in 1904, and feeling that the sinus might possibly be drained (gravity method) by way of the sphenoidal cavity through the same side of the nose. J. C. Beck, in 1912, in investigating the subject of approaching the hypophysis from various routes, developed a method of reaching the hypophysis by way of the antrum following the Jansen operation, and operated successfully two cases of hypophyseal tumor. From cadaver work, having in mind also at the same time the cavernous sinus, both Beck and J. R. Fletcher emphasize the tremendous difficulty of reaching this sinus through the nose. At best, certainly any kind of an operation will many times be seen to be a useless proposition under some conditions.

(4) ARTERIOVENOUS ANEURISM (RUPTURE OF INTERNAL CAROTID ARTERY INTO CAVERNOUS SINUS).

Fifteen cases of rupture of the internal carotid artery in the cavernous sinus, producing arteriovenous aneurism with pulsating exophthalmos, for which ligation of the common carotid or internal carotid became necessary, were reported by letter. Improvement or full recovery ensued in twelve, death in three cases. The principal causes as given were syphilitic weakening of the wall of the artery and traumatism as by fracture or a blow on the head. In one of these cases (Halstead) thrombosis of the cavernous sinus took place after ligating the internal carotid artery with, however, good recovery from the cavernous sinus thrombosis. (Table 4.) Rupture of the carotid into the cavernous has been extensively reviewed in literature the past few years.

(5) INJURY TO CAVERNOUS SINUS DURING MISCELLANEOUS OPERATIONS.

Three cases were reported by letter in which the cavernous sinus was quite badly injured—two during a Gasserian ganglion operation with, however, good recovery, and one during an hypophysis operation (Halstead), with death from intracranial hemorrhage. In the literature appended, Emerson[62] reports serious injury of the sinus with death from curettement of the sphenoidal sinus. Cushing[82] reports a case of injury to the sinus during a Gasserian ganglion operation, followed by cavernous sinus thrombosis and recovery. The attention which the pituitary body is receiving, with operative attempts at treatment, must surely in time throw much more positive light on the cavernous sinus and its transient structures.

(6) MALIGNANT GROWTHS INVOLVING CAVERNOUS SINUS.

Two cases of sarcoma with death (Benjamin) were reported in my list of question-letters, but reported without any further data. In literature appended, Knapp[105] reports a case of orbital sarcoma followed by aseptic thrombosis of the cavernous sinus, with successful sinus operation by Hartley, in which the patient lingered for over two months. Macewen[102] mentions a case of septic thrombosis and death from carcinomatous involvement.

(7) CONGENITAL AFFECTIONS.

One congenital growth (fibrocystic) was reported by Veer (Table 7), in which the zygoma and malar bones were resected in order to reach the area, and many vessels surrounding the cavernous litigated. The patient made a good recovery. I have not obtained as yet a detailed report of this case. One might well expect here also the possibility of some of the rarer forms of congenital angiomata deep within the orbit, in which a correct diagnosis is difficult, to involve the cavernous sinus in some of its ramifications.

(8) MARASMIC (ASEPTIC) THROMBOSIS.

No cases of purely marasmic thrombosis were reported in the question-letters sent out. A few instances are on record in literature, however, occurring during the course of debilitating or marasmic diseases, such as pregnancy, typhoid fever, of socalled marasmic thrombosis of this vessel. Zentmayer-Weisenburg[73] report such a case associated with menstrual disturbances and pregnancy, and Knapp[105] presents a case of aseptic thrombosis from sarcoma of the body of the sphenoid bone and apex of the orbit originating in the cavernous sinus.

(9) INTERMITTENT EXOPHTHALMOS.

This is a most interesting condition, new, I think, to most of us, and its exact relationship to the cavernous sinus, if any, still a little unsettled. For the present, as mentioned, it seems permissible to mention it here.

No cases of this affection were mentioned in the question-letters, so that the literature of the subject had to be consulted for the information. True intermittent exophthalmos, a rare condition, is fully reviewed by Posey[80] and Alger,[75] and must not be confused with pulsating exophthalmos, which last is due to rupture of the internal carotid artery in the cavernous sinus.

In a case of true intermittent exophthalmos, owing to changes in the orbital veins, one eye will be pushed forward out of the orbit, but quickly recedes as soon as venous pressure in the orbital veins and cavernous sinus becomes normal. In a typical case the eye proptoses in a few seconds whenever the head is in a dependent position, as in stooping, or whenever a venous congestion of the cranium is produced by a great muscular exertion or by a local pressure on the neck. The explanation of the phenomenon seems to be that under certain conditions which produce increased muscular tension in a very tortuous ophthalmic vein, a sudden kink or constriction results, completely stopping the return of blood from the orbit to the cavernous sinus. As soon, however, as the venous pressure is lowered in the cavernous by a change of posture or relief from muscular strain, the kink or bend is relieved from pressure posteriorly, the vein emptied and the eye sinks back into its natural position in the socket. As there seems to be no record of an autopsy on a case of intermittent exophthalmos, our knowledge of this pathologic condition is largely theoretic.

In concluding this paper and especially from a study of the many artices comprising the index it is difficult to refrain from commenting on the gradual signs of progress being made year by year with reference to our better understanding of the anatomy, pathology and diagnosis of affection of the cavernous sinus.

TABLE 1

Septic Thrombosis of Cavernous Sinus

Doctor submitting information	Diagnosis	Etiology	Treatment	Result
Collins, A. N. Duluth, Minn.	Septic thrombosis	Purulent disease of nasal sinuses	Frontal sinus opened and drained	Death
Strout, E. Minneapolis, Minn.	Septic thrombosis	Accessory sinuses	Not given	Death
Hitz, H. B. Milwaukee, Wis.	Septic thrombosis	Not given	Not given	Death
Blair, V. P. St. Louis, Mo.	Septic thrombosis	Suppurative ethmoiditis	Accessory cavities drained	Death
Oppenheimer, S. New York City	(1) Septic thrombosis (2) Septic thrombosis	Aural Aural	Not given Not given	Death Death
Sudler, M. T. Lawrence, Kan.	Septic thrombosis	Aural	Not given	Death
Patton, J. M. Omaha, Neb.	Septic thrombosis	Boil of neck	Not given	Death
Geiger, Jacob St. Louis, Mo.	(1) Septic thrombosis (2) Septic thrombosis (3) Septic thrombosis	Chronic ear discharge Aural Aural	Not given Not given Not given	Death Death Death
Witherspoon, T. C. Butte, Mont.	Septic thrombosis	Typhoid fever—no details	Not given	Death
Lilienthal, H. New York City	(1) Septic thrombosis (2) Septic thrombosis (3) Septic thrombosis	Not given Osteomyelitis lower jaw Furuncle vestibule of nose	Exploratory craniotomy Not given Not given	Death Death Death

Rixford, E. San Francisco, Cal.	Septic thrombosis	Ascending infection pterygoid muscle and base of skull	Not given	Death
Gifford, H. Omaha, Neb.	Septic thrombosis	Aural	Not given	Death
Cott, G. F. Buffalo, N. Y.	Septic thrombosis	Aural	Not given	Death
Hamann, C. A. Cleveland, Ohio	(1) Septic thrombosis (2) Septic thrombosis	Peridental abscess Frontal and maxillary infection	Not given Not given	Death Death
Lynch, R. C. New Orleans, La.	(1) Septic thrombosis (2) Septic thrombosis	Aural Aural	Not given Not given	Death Death
Raymond, Alfred Seattle, Wash.	(1) Septic thrombosis (2) Septic thrombosis	Accessory sinuses Acute tonsilitis	Not given Not given	Death Death
Kyle, J. J. Los Angeles, Cal.	Septic thrombosis	Aural	Not given	Death
Jepson, Wm. Sioux City, .owa	Septic thrombosis	Accessory sinus disease	Not given	Death
Scholz, R. P. St. Louis, Mo.	Septic thrombosis	Aural	Radical mastoid and ligation lateral sinus	Recovery
Kanavel, A. B. Chicago, Ill.	Septic thrombosis	Neck infection	Local neck drainage	Recovery
Halstead, A. E. Chicago, Ill.	Septic thrombosis	Ethmoiditis and syphilis following ethmoid operation	Expectant treatment	Recovery

TABLE 2

Gun Shot and Penetrating Wounds of Cavernous Sinus

Doctor submitting information	Diagnosis	Treatment	Result
Babler, E. A. St. Louis, Mo.	1—Gun shot wound 2—Gun shot wound	No operation Not given	Death Death
Yates, J. L. Milwaukee, Wis.	Gun shot laceration		Death
Basham, D. W. Wichita, Kan.	Gun shot wound		Death
Raymond, Alfred Seattle, Wash.	1—Gun shot wound 2—Injury, right optic nerve and sinus torn across. Frontal lobe destroyed	Not given Tamponade	Death Recovery
Halstead, A. E. Chicago, Ill.	1—Gun shot wound through ear, lodging in sinus cavity. Developed sinus thrombosis and brain abscess. Sinus operated by Gasserian ganglion route and bullet removed. 2—Gun shot wound		Death Recovery, loss of both eyes

TABLE 3

Involvement From Fracture of Skull

Doctor submitting information	Diagnosis	Treatment	Result
Babler, E. A. St. Louis, Mo.	Fracture of base with rupture cavernous sinus	Not given	Death
Ochsner, A. J. Chicago, Ill.	Compound fracture with laceration cavernous sinus	Cavernous sinus tamponed	Recovery in ten days
Pearse, H. E. Kansas City, Mo.	Fracture base with laceration sinus wall	Operative removal of clot	Death—meningitis
Basham, D. W. Wichita, Kan.	Fracture with injury to cavernous sinus	Not given	Death
Bevan, A. D. Chicago, Ill.	1—Fracture of base causing cavernous sinus thrombosis 2—Fracture of base causing cavernous sinus thrombosis	Not given Not given	Death Recovery—loss of eye

TABLE 4

Rupture Internal Carotid Artery in Cavernous Sinus Arteriovenous Aneurism

Doctor submitting information	Diagnosis	Etiology	Treatment	Result
Schwyzer, A. St. Paul, Minn.	Pulsating exophthalmos—arteriovenous aneurism	Not given	Ligation common carotid	Cured now six years
Dennis, W. A. St. Paul, Minn.	Pulsating exophthalmos	Fracture base of skull	Ligation common carotid and ophthalmic vein	Relief, but not complete
Vaughan, G. T.	Arteriovenous aneurism	Not definitely ascertained	Ligation right internal carotid	Death
Martin, Edward Philadelphia, Pa.	1—Arteriovenous aneurism 2—Arteriovenous aneurism	Traumatic	Ligation orbital veins and carotid Not given	Relieved Not given
Lilienthal, H. New York City	Pulsating exophthalmos	Followed mastoid operation	Ligation both common carotids	Death
Griffith, J. D. Kansas City, Mo.	Arteriovenous aneurism	Not given	Ligation common carotid	Death
Dean, L. W. Iowa City, Iowa	Pulsating exophthalmos	Shot through orbit	Common carotid ligated	Improved
Bevan, A. D. Chicago, Ill.	Six cases aneurism internal carotid in cavernous sinus	Not given	Ligation common carotid	Recovery or very much improved
Halstead, A. E. Chicago, Ill.	Arteriovenous aneurism and thrombosis of cavernous sinus	Thrombosis followed ligation of carotid	Ligation internal carotid	Recovery from cavernous sinus thrombosis

TABLE 5

Injury to Cavernous Sinus During Miscellaneous Operations

Doctor submitting information	Diagnosis	Treatment	Result
Harris, M. L. Chicago, Ill.	During Gasserian ganglion operation	Tamponade controlling the profuse hemorrhage	Good; recovery
Davis, G. G. Philadelphia, Pa.	During Gasserian ganglion operation	Packing	Recovery
Halstead, A. E. Chicago, Ill.	During an hypophysis operation	Not given	Death from intracranial hemorrhage

TABLE 6

Malignant Growths Involving Cavernous Sinus

Doctor submitting information	Diagnosis	Treatment	Result
Benjamin, A. E. Minneapolis, Minn.	1—Sarcoma 2—Sarcoma	Details not given Details not given	Death Death

TABLE 7

Congenital Affections of Cavernous Sinus

Doctor submitting information	Diagnosis	Treatment	Result
Vanderveer, A. Albany, N. Y.	Congenital growth Fibrocystic	Resection zygoma and malar bone. Ligation of many veins surrounding cavernous	Good recovery

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XXIX.

A CASE OF VAGOTONIA, APPARENTLY ORIGINATING IN THE NASAL ACCESSORY SINUSES.*

By George Fetterolf, M. D., Sc. D.,

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Vagotonia can be defined briefly as a condition of excitement or hypertonus of a group of nerves called the "extended vagus." The phrase "extended vagus" is applied to a nerve series which includes not only the pneumogastric, but also a group of nerves which functionate similarly to it. Antagonistic to the vagus group is the sympathetic, and there exists a condition of it called sympatheticotonia, which can be defined as a state of excitement or hypertonus of the sympathetic system. In order to locate accurately the "extended vagus" and sympathetic systems, a brief review of a few points in the anatomy of the nervous system might be in order.

Connected with the brain and spinal cord is a long series of paired nerves, beginning above with the first cranial and ending below with the last coccygeal. The fibers of these are divided into two groups, the animal and the vegetative. The animal group consists of sensory and motor fibers, and supplies the organs of sensation and the voluntary muscles.

The vegetative system supplies all those glands which have ducts, and all involuntary muscle tissue, including that of the muscular organs.

It has been found that this vegetative system consists of two distinct parts, called the autonomic system and the sympathetic system. These two cannot be separated by dissection, but through various tests, especially drug tests, their individuality has been established beyond question.

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*Read before the American Laryngological Association, at the thirty-eighth annual meeting, Washington, D. C., May 9, 10 and 11, 1916.

The place of origin of the vegetative fibers is the brain and the spinal cord. The sympathetic portion arises from the cord alone, beginning at the second thoracic nerve and continuing down to and including the second lumbar.

The fibers which comprise the autonomic portion arise in part from the brain stem and in part from the sacral region of the cord.

The distribution of these fibers is widespread, but only that of the autonomic system will be given, as it is the one with which this contribution mainly is concerned. On account of direct clinical bearing, their distribution will be described in some detail.

The fibers which arise from the brain are contained in the third, seventh, ninth, tenth and eleventh cranial nerves.

Those of the third nerve go to the ciliary ganglion, whence other neurones take up the impulse and carry them to the ciliary muscle and the sphincter of the iris. It is probable, too, that the elevator of the upper eyelid receives autonomic fibers.

The fibers of the seventh nerve are those added to it by the pars intermedia of Wrisberg, and with those from the ninth, tenth and eleventh, go to sundry ganglia, whence secondary or postganglionic fibers emerge. These "innervate the blood vessels of the mucous membrane of the mouth, throat, nose, and paranasal sinuses, the salivary glands, the heart muscle, the glands and musculature of the trachea and bronchi, the glands and musculature of the digestive tube from the esophagus to the colon (gullet, stomach, intestine, liver and biliary passages, pancreas, and ducts), as well as the blood vessels and tubules of the kidney."[1]

The sacral part of the autonomic system derives its fibers from the first, second, and third sacral nerves, and is distributed through the pelvic plexus. It supplies fibers to the "musculature and glands of the lower colon, sigmoid, rectum, anus, urinary bladder, and genital organs, and to the arteries of the rectum, anus, and internal and external genitals."[1]

In action the two groups, the autonomic and the sympathetic, are antagonistic, and it is generally conceded that all glands which possess ducts and all involuntary musculature receive a supply from both. Upon the maintenance of a proper balance between the two depends the normal functionating of the structures to which they go. If the autonomic supply is irritable, the organ will overfunctionate in one direction, and if the sympathetic is in a condition of hypertonus or excitement, the excess of action will be in the other. As glands and muscles are the organs to which these impulses go, the result will be manifested by hyper- or hyposecretion on the one hand, or by spasm or relaxation on the other.

The control of the entire vegetative systems, both autonomic and sympathetic, is believed to lie in the cerebrospinal axis, where possibly a regulating center exists, and in the glands of internal secretion, the socalled endocrinous glands. This latter is quite well established for the sympathetic system, adrenin having been shown to act generally as a stimulator to the entire distribution of this group. For the autonomic system no drug has been found which acts uniformly upon all parts of it. The nearest approach to such a drug is atropin, which is a sedative to practically the entire autonomic system, dilating the pupil, checking the flow of saliva and sweat, and relaxing contracted involuntary muscle. Eppinger and Hess[2] state that it is selective, in that the sacral part of the autonomic system is practically unaffected by it. In a recent paper, Block[3] takes an opposite view, as the result of having used atropin in what he calls the "vagotonic type" of dysmenorrhea, substituting this term for the older "essential or uterine dysmenorrhea." In this form of the disease he has used atropin in a limited number of cases with "most satisfactory results." Even more selective is pilocarpin, which has a powerful effect upon the salivary and sweat glands, producing oversecretion, and hence clearly to be regarded as a stimulator of part of this system.

Pilocarpin is known to be so reliable and constant a stimulator of the autonomic system that it is used in testing for the presence of vagotonia. One-twentieth to one-sixth grain is given hypodermically, and if the test is positive there will be noted salivation, lacrimation, sweating, cardiorespiratory arhythmia and hyperperistalsis, all these out of proportion to the size of the dose. Writers on this subject believe the drug tests to be quite important. The definition of vagotonia given by Eppinger and Hess[2] is as follows: "A functional, autonomic system disease, regarded so for the reason that all its symptoms may be identified with those of a state of stimulation of the extended vagus or autonomic nervous system." But they do not consider this definition to be complete unless there is added to it "a hypersensitiveness to pilocarpin"[1] and "a relative decrease of reactivity to sympathetic stimuli."[2]

The symptoms of vagotonia in general are such as would be expected when the autonomic system is stimulated, and they represent activity of function at the terminal distribution of the nerves concerned. There are many of these symptoms, but just a few will be mentioned to indicate their general nature.

Cramp of the ciliary muscle, spasm of accommodation and widening of the palpebral fissure are examples of what would result from hypertonus of the fibers from the third nerve.

Salivation and congestion of and hypersecretion from the nose and nasopharynx would follow irritation transmitted through the otic, sphenopalatine and submaxillary ganglia.

To the vagus itself can be ascribed many phenomena, such as bronchial asthma, bronchial hypersecretion, laryngeal crises, bradycardia, gastric crises, hyperperistalsis and excessive gastric and intestinal secretion.

Through the sacral part of the autonomic system could pass impulses which would give rise to such conditions as spasm of the anal sphincter, hypersecretion from the intestine, dysmenorrhea, etc.

The blood picture shows an excess of eosinophiles. Hopkins[4] says: "Eosinophilia occurs especially where the skin, lung or intestinal element is present, and is increased after pilocarpin, often markedly."

An interesting observation by Eppinger and Hess[2] is that in vagotonics there is likely to be a marked anesthesia of the entrance to the pharynx, a condition usually ascribed to hysteria. These authors say: "We wish to state that, on the basis of our observations, we regard this phenomenon as entirely unrelated to hysteria."

The treatment of the condition is the same as that of any other secondary disease, viz., find, if possible, and eliminate a cause or focus, build up the health and give proper medication. The last factor, the medicinal treatment, can be managed on one of two principles, either administering a sedative for the autonomic system, or overcoming the tonus of the latter by giving a stimulator to its antagonist, the sympathetic system. This principle is illustrated notably by the successful treatment of spasmodic bronchial asthma, success being possible either by quieting the autonomic system with atropin or by stimulating the sympathetic by the local or hypodermic use of adrenin.

The case I wish to report in this connection was referred to me by Dr. Alfred Stengel, who had given it long and careful study. The patient was a lad of about twelve years, who for five years had gradually failed in health, with an intermission or two of slight improvement. He presented many symptoms of autonomic tonus and, in addition, had evident disease of his paranasal sinuses.

His impaired health dated back to an attack of measles at the age of seven, which left him with marked nasal discharge and expectoration, which have persisted ever since. There was at times a loose cough, which was productive of a thin, mucoid sputum, which never became yellow nor blood tinged. For a year there was profuse sweating at night, and his sleep was much disturbed by sneezing, coughing and expectorating. His adenoids were removed during this time, but the result was unfortunate, as immediately afterward his nasal discharge increased and he developed abscesses in both ears.

Three years after the appearance of the first symptoms Dr. Stengel saw him. No cause for his symptoms was found, physical examination being negative. He was tested with pilocarpin for vagotonia by Dr. Hopkins, and the result was p tive. He was put upon guaiacol carbonate and extract of be donna, and some slight improvement resulted.

Two years later he returned to Dr. Stengel, a very sick li boy, having lost nine pounds in four weeks. At this time he was vomiting all his meals, a condition which soon changed to a weekly attack of severe abdominal pain, nausea and vomiting, which would last about two days. The vomitus was profuse and consisted almost entirely of mucus. With this there was profuse salivation, of a clear, watery character. Soon was added to these conditions frequent watery bowel movements. Medication was of little value, and the boy's condition was becoming a desperate one.

It was at this time I was requested to examine him, and aided by an X-ray examination by Dr. H. K. Pancoast, a diagnosis of chronic suppuration in the posterior ethmoidal and sphenoidal cells of the left side readily was made. The surgical indications were clear, and I removed the posterior end of the middle turbinated and opened widely the offending cells.

Improvement was immediate and very striking, as almost at once his symptoms disappeared. In two months he gained ten pounds and was a perfectly well boy except for some discharge from his nose. Later Dr. Herbert Fox administered autogenous vaccines, and this was followed by almost total disappearance of the nasal mucus.

Our interpretation of the case is this: The boy was one with a very unstable vegetative nervous system, and it has been observed that in such a state small stimuli can produce large reactions. The irritation which would cause such a condition of autonomic hypertonus may (quoting Hopkins[4]) "arise from noxa in the form of bacterial toxins, as during or after acute infections, from drugs or from the products of deranged metabolism, mechanical irritation and so forth." There was present in this case a primary acute infection, measles, followed by prolonged chronic suppurative sinus disease, and we believe that the consequent toxemia and irritation were the exciting factors. For, when this was relieved, the widespread vagotonic symptoms disappeared so promptly as practically to bar accidental coincidence.

As far as I have been able to discover, this case is the first to be placed on record in which such a distinct entity as chronic sinus disease was presumably the exciting cause of such widespread vagotonic manifestations, and in which prompt disappearance of these symptoms followed relief of the local condition. By the free drainage of suppurating sinuses this patient was almost instantaneously relieved of salivation, cough, expectoration, severe abdominal pain, vomiting and diarrhea, for none of which symptoms a physical cause could be found.

As a sinus case per se there was nothing notable about it, as the diagnosis was easy and the treatment simple. The big feature is the field of observation and deduction such a case opens up. It does seem that many other conditions which are but vaguely understood may possibly become clarified when viewed as vagotonic or sympatheticotonic phenomena. For example:

The treatment of bronchial asthma with adrenin or with atropin has been successful in many instances.

Noteworthy has been the nasal treatment by our colleague Emil Mayer,[5] Brettauer[6] and others, of selected cases of dysmenorrhea.

Another fellow member, Greenfield Sluder,[7] after painstaking study, has succeeded in relieving certain symptoms by treating the sphenopalatine ganglion.

These are but a few of the conditions upon which some light may be shed. Undoubtedly many others will suggest themselves as being based upon a derangement of visceral nerve supply. Should such prove to be the case, they will constitute a new group whose pathology can be described in definite terms.

In conclusion. An obvious criticism which may be and has been made of this entire subject is that there is much of the speculative in it. In answer to this comment, Barker[1] says: "We find out new facts by forming hypotheses. No man works without an hypothesis. So long as we use hypotheses only to stimulate work, to clarify our views, or to hold even temporarily widely separated facts together, I think they may be most helpful."

Eppinger and Hess,[2] with the same thought in mind, say: "We know full well that many of the things which we have sated stand but on unsteady ground, but, in spite of this, we hope to have given the impetus to further investigations which may be of great significance for special pathology and therapy."

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XXX.

A CASE OF SEPTIC THROMBOSIS OF THE JUGULAR BULB, WITH REPEATED FORMATION OF SEPTIC THROMBI IN THE SIGMOID AND LATERAL SINUSES. WITH REFERENCE TO THE LITERATURE ON INVOLVEMENT OF THE TORCULAR HEROPHILI IN SUCH CASES.*

By John Randolph Page, M. D.,

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The proximity to the torcular Herophili of the final thrombus which formed in this case, and the cause of its limitation at this point, have brought to mind some conflicting views regarding the tendency of a thrombus in this region to extend across the torcular to the opposite side. The discussion of this subject by Mr. Ballance and Mr. H. E. Jones, senior surgeon of the Liverpool Eye and Ear Infirmary, will be referred to later.

A delicate girl, twelve years of age, developed an abscess in her right middle ear, upon which a myringotomy was performed the following day. The third day after the myringotomy was performed she had a chill, and her temperature rose to 103.8° by mouth.

The following day I saw her and advised immediate mastoidectomy. Nothing unusual was found beyond a well infected mastoid, but because of the chill on the previous day and the septic appearance of the child, a guarded prognosis was given, and the precaution was taken to remove the plate over the vertical limb of the sinus for inspection of its wall. It was apparently normal, but the possibility of infection being in the bulb was remarked at the time.

The patient, being operated on out of town, was left in the care of Dr. Kellogg, of Norwalk, Connecticut, who reported the following morning that the temperature had gone to 106.5° during the night, but that the condition was then good, temperature at noon being 99.8°. In the afternoon, after a chill, the temperature rose to 107.5°.

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Exploration of the sigmoid sinus that night revealed scant bleeding from the bulb, but no clot in the vein, and no apparent change in its wall. The internal jugular was resected and the lower portion of the sigmoid sinus wall cut away.

A fall in the temperature to 98°, accompanied by an alarming collapse, followed three hours after the jugular resection. Next day, however, the child's condition was fair and spirits good, but the following night the temperature rose to 107.2°, and the next night to 105.5°. After this her condition improved, but at the end of a week signs of sepsis began to return.

On the twelfth day after the jugular resection a large abscess was opened in the upper part of the vertical limb of the sinus. The sinus wall was excised beyond the clot, and free bleeding was obtained. Culture from the pus showed streptococcus hemolyticus; blood culture was negative—leucocytes 20,000, ninety-two per cent polynuclears. She now complained of blurred vision. A distinct double optic neuritis was present, more marked on the right side. The disturbance of vision apparently subsided in a few days, and no further ophthalmoscopic examinations were made.

Immediate improvement followed this operation, but within a week a return of the high temperature and sepsis made further exploration of the sinus in the direction of the torcular necessary.

Because of the patient's extreme weakness the operation had to be performed in bed without an anesthetic. More bone was removed and the sinus further exposed for about two centimeters. Incision of it released another dram of thick gray pus from its interior. Gentle removal of the torcular end of the thrombus was made with forceps, and a good flow of blood was obtained which flushed out the vein. Iodoform gauze was then introduced into the lumen of the vein for one inch or more, with the hope that the clot might be induced to form beyond the area of infected wall. This was followed by marked improvement for six days. On the seventh day signs of sepsis reappeared, and a fourth exploration of the sinus still further back was necessary.

At this operation (under ether) an abscess, similar to the other two, was found, the sinus being filled with dark gray pus, which extended back to within three centimeters of the posterior occipital protuberance. There were no granulations on the wall, but it had an infected appearance. The clot in the vein, which extended beyond this point, was removed with forceps, and the bleeding which followed its removal was controlled by my finger in the vein. With my finger in the vessel for two or more centimeters I could feel a septum which seemed to divide its lumen at the torcular.

Feeling that the vein wall might be infected to this point, I inserted iodoform gauze as far back as my finger reached, and later felt fearful lest it might produce a thrombus in the other sinuses. This gauze was removed on the second day, and its removal was followed by a surprisingly small flow of blood, due probably to a partial thrombosis of the superior longitudinal and a naturally small communication with the sinus of the opposite side.

During the progress of the case, which after this operation made an uninterrupted recovery, Dr. John D. Richards expressed the opinion that the thrombus would not extend to the opposite sinuses of the torcular, as the rapid flow of blood through them at this point would tend to limit the formation of the clot.

In this opinion he is supported by Mr. Ballance,[1] who, in 1890, made the following statement: "It is an error to suppose that the lateral sinuses are continuous at the torcular Herophili; the right sinus is continuous with the superior longitudinal sinus, and the left sinus is, as a rule, a continuation of the straight. There are sharp bends in the venous channels where the lateral sinus commences, and these are joined across the front of the internal occipital protuberance by means of a separate sinus, or cross branch, of small size; thus a thrombosis of one lateral sinus need not, and probably will not, be accompanied by thrombosis of the other lateral sinus, and so life is spared." While the above arrangement is not invariable, it will be found to be the general rule.

Cunningham states[2] that "the superior sagittal sinus turns to the right and becomes continuous with the right transverse sinus, while the posterior end of the straight sinus turns to the left and joins the left transverse sinus. In a certain number of cases this arrangement is reversed, and not uncommonly there is a communication between the right and left transverse sinus across the front of the internal occipital protuberance." He then says that "occasionally the superior sagittal, the two transverse sinuses, the straight sinus, and the occipital sinus unite anterior to the occipital protuberance in a common dilatation—the 'confluens sinuum.'"

Despite the fact that this last arrangement is the most infrequent of any, it seems, from the descriptions given of operations on the transverse sinus in this region, the one thought to be most common. This very general impression has been due to the meager descriptions of the textbooks on anatomy, which refer to the torcular Herophili only as a marked dilatation of "irregular form" where the five sinuses meet.

In regard to the complete separation of the two sides Dwight[3] says, in a discussion of the subject, that he "cannot agree with Rüdinger, that complete separation of the two parts of the torcular is common, this and perfect symmetry being about equally rare." The right lateral sinus is usually considerably larger than the left, and the asymmetry may extend to complete absence of one lateral sinus and jugular vein, as has been referred to by Eagleton[4] and others.

Hugh E. Jones,[5] senior surgeon of the Liverpool Eye and Ear Infirmary, is of the opinion, contrary to Mr. Ballance, that "once at the torcular the progress of infection to the other great sinuses is easy," and that the question of extension of infection to the opposite sinus has not received the attention it deserves in literature. He supports this opinion by reference to a case reported by himself, a remarkable one by Whitehead,[6] and one by Lodge; but with the exception of Whitehead's case, the others appear to me not to have been due to gradual extension of the thrombus from one sinus to that of the opposite side, but to the formation of a clot shortly before death in the whole region of the torcular, due, in all probability, to the general condition of the patient rather than to a true extension of the septic thrombus from the lateral sinus; for while thrombosis of the whole region of the torcular and all the sinuses has been repeatedly reported in cases profoundly septic and debilitated from various causes, so far as I have been able to ascertain the literature records few cases in which a truly septic thrombus extended from the mastoid region of one side across the torcular to the opposite side.

The extension would naturally take place only to that sinus of which the one involved was a continuation, and because of the usual separation, except for the rather variable communication between the two sides, it would not be likely to extend to the other side.

An example of this is prettily demonstrated in a case reported by Dr. Simpson,[7] in which the mastoid and lateral sinus of the left side had been operated on. Autopsy showed "the superior and inferior longitudinal sinus to be patent and without change, the left lateral sinus completely occluded, a thrombus extending from the point of operation at the left mastoid to the torcular Herophili and also to a slight extent into the straight sinus. The right lateral sinus was quite free and healthy."

In the case referred to by Mr. Jones[8] the autopsy report says: "From the junction of the superior petrosal sinus with the lateral sinus up to within one-half inch of the torcular Herophili, the walls of the sinus were healthy, but here commenced a patch of phlebitis one inch long, etc., etc, which extended across the midline to the superior longitudinal sinus and right lateral sinus." It will be seen from this that there was not an extension from the original thrombus in the sigmoid, but that one formed spontaneously in the torcular, due probably to the extremely septic and weakened condition of the patient.

In another article,[8] in opposition to Mr. Ballance, he cites the twenty-two cases of lateral sinus thrombosis associated with ear diseases, collected by Newton Pitt,[9] in which the clot extended to the longitudinal sinus in four, to the opposite lateral in three, and in one to all the sinuses, and says that "not only, therefore, is it a not unlikely occurrence, but a comparatively common one." Again we think his conclusion unjustified, for it will be seen from this collection of Pitt's that in four cases the clot extended to the longitudinal sinus (a continuation of the right lateral), and not to the opposite side; so while this leaves four to be accounted for, it cuts down his percentage by half. And among these we are not convinced that there were not some similar to the one he reported in which the thrombosis was due to general causes rather than to a real extension from the primary thrombus in the mastoid region.

If I may be permitted to cite one more reference, Lidell,[10] in 1874, collected thirteen cases of sinus thrombosis associated with mastoiditis in which autopsies were performed, and in one of these there was extension a short distance into the opposite side. The report says: "A light colored, purulent looking plug distended the right lateral sinus and adhered closely to its walls—the same clot filled the torcular Herophili also, and extended a little way into the left lateral and straight sinuses. The posterior two-thirds of the superior longitudinal sinus was filled with greenish pus, which also extended to the veins coming from the pia on the right side."

This report, to my mind, supports Mr. Ballance rather than Mr. Jones, for the superior longitudinal sinus was, no doubt, continuous with the right lateral, in which the trouble began, and two-thirds of its length became involved before the torcular was crossed and the opposite lateral and straight sinus were involved, even for "a little way."

In Whitehead's very exceptional case, which recovered, there was no doubt of the involvement of both sides of the torcular.

After operation on the right mastoid he says: "The sinus was freely exposed and found to be thrombosed; the overlying bone was removed back to and over the torcular Herophili and the sinus laid open the whole distance; the openings of the longitudinal sinus and of the lateral sinus of the other side were blocked with firm clot, and a small curette passed into the opposite sinus failed to set up bleeding." Such cases are very rare.

While we not infrequently hear of a thrombus being encountered which extends from the torcular to the bulb, and occasionally a case in which no bleeding is obtained from the torcular region, no minute descriptions are given of the operations in this region, and I think it will prove interesting to observe as often as possible in the living, and at autopsies, just what direction and to what extent lateral sinus thrombosis progresses in this region. It will be interesting also to note more carefully in future the variations in the anatomy of this interesting meeting place of the sinuses.

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XXXI.

MALARIA SIMULATING DISEASES OF THE MASTOID AND FRONTAL SINUS.*

By Charles W. Richardson, M. D.,

Washington.

Malaria, like syphilis, affects many organs and tissues of the body, producing lesions thereby, and through action upon the nervous system may simulate diseases of other organs and tissues which are not directly affected by the parasites of these diseases.

I wish to present to you today two interesting cases wherein the malarial parasite, through its invasion of the organism, produced striking subjective evidences in one case of mastoiditis, and in the other of frontal sinuitis. Beyond these interesting facts, these cases present nothing of unusual interest. The diagnoses were readily made through exclusion, without any difficulty, and were confirmed by the blood examinations.

The first case, that of simulating mastoiditis, was brought to my attention by my assistant, Dr. W. H. Huntington, immediately after I had finished a rather laborious afternoon in the operating room. The provisional diagnosis was mastoiditis, with probably intracranial complications. Due to lack of time, I made only a cursory examination, and quickly reviewing the history, found the evidence such as to warrant a strong doubt in my mind both as to the mastoid infection as well as the more serious phase of the case. I ordered a blood examination and count and urinalysis. Dr. Huntington had already made an examination of the eye grounds. In the late afternoon I made a most thorough examination of the patient, and through the history and the results of this examination I became convinced that the mastoid symptoms were due to other causes than sup-

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***Read before the American Otological Society, May 10, 1916.**

puration. At this time I had also the benefit of both the blood examination and the urinalysis.

The history of the case, as prepared by Dr. Huntington, is as follows:

Case 1.—Miss M. L. H., female, white, twenty-one years of age, was admitted to the Episcopal Eye, Ear and Throat Hospital, December 8, 1915. Family history negative.

Previous History.—Had measles when eight years of age. Had chills during infancy. No previous affection of the ears.

Present Illness.—Marked evidence of great prostration, requiring support in walking. Very pale, Had acute suppurative otitis August 15, 1915, with spontaneous rupture of membrana tympani. Discharge continued until November 6, 1915, when it ceased. At this time postauricular pain became manifest and gradually grew worse, with swelling back of ear and in neck. About a week later headaches, which were not constant and confined to occipital region. Vomited at times. About second week in November had chills, which were unattended with sweats. Complained of great thirst and drank abundance of water. Urine at that time was scanty and very dark. During past few days has had marked pain over mastoid region. For past two weeks has noticed gradual diminution of vision.

Physical Examination.—Left ear normal. Right ear shows a central kidney-shape perforation. Ear dry. Pressure over mastoid, as well as along course of jugular vein, causes pain. No edema. Pulse 120. Temperature 101°.

Eye.—Slight exophthalmos. Pupils widely dilated, but reacted to light and accommodation. No nystagmus. Vision impaired. Figures at ten feet. Eye ground shows bilateral papillitis, with hemorrhage and exudate around vessels. Arteries small. Veins congested and tortuous. No exudate at macular region.

When I first saw this patient I was impressed with her hysteric facial expressions and marked pallor. Examination of ears demonstrated them to be as given in the history. The tenderness over the right mastoid was very great, as well as along the course of the jugular vein. When the patient was placed upon her feet and told to walk, she would advance a few steps and then begin to totter, showing rather an evidence of weakness than of any central lesion. The double optic papillitis also excited my suspicion, as there were no evidences of any acute cerebral lesion that could account for its centric origin. The head pain, the pallor and the slight mental hebetube inclined me to await an examination of the urine and blood before making a diagnosis.

The urinalysis made from urine drawn by catheter was as follows: Color, dark brown, very turbid. Specific gravity 1023. Reaction, acid. Sugar, considerable quantity. Albumin, trace. Microscopic: Casts, granular, hyaline and epithelial, with a predominance of granular. Many disintegrated blood cells.

Blood Examination.—Malarial parasites present. Leucocytes, 8,800.

The patient was given quinin in large doses, large draughts of water, the Murphy drip and hot packs. Under this treatment the temperature gradually subsided from 101.2° on entrance to 99°. On December 11th there was a rise in temperature to 102°, unattended with chill, which subsided to normal December 12th. From December 12th until the patient was discharged the temperature was flat. Blood examination, made after December 12th, demonstrated a freedom from malaria. The urinalysis showed normal urine after December 16th. The mastoid tenderness and the general condition of the patient improved from day to day. The papillitis improved considerably, and the vision became clearer, but neither had returned to the normal when the patient was discharged from my service.

January 4, 1916, some weeks later, the patient returned to the hospital on account of the want of further improvement of the eye condition. On account of a brother of the patient, an attendant in the dispenary service, having been found to be a syphilitic, a Wassermann was made upon the patient with a positive finding.

Case 2.—The case of frontal sinuitis presents a more clearly defined history of malarial origin than the case of mastoiditis, as it was uncomplicated. The patient was a young locomotive fireman, who lived in a malarial district in the State of Maryland. When I first examined him he manifested all the subjective evidences of a frontal sinus infection. He complained of intense frontal head pain. There was a marked tenderness over the left frontal region, which was particularly marked in the floor of the sinus. Inspection of the nose demonstrated an absolutely normal nasal chamber. Postnasal examination also showed no deviation from the normal. The following day I again went carefully over the patient, and from the total absence of all objective evidences of frontal sinus infection, and from the fact that the patient had dwelt in a malarial district, I directed that a blood examination and count be made. The patient was admitted to the Episcopal Eye, Ear and Throat Hospital, December 28, 1915. The history is as follows:

J. W. S., twenty-four years of age, living in Seat Pleasant Heights, Maryland. Mother, living, well; age, forty-seven years. Father, living, well; age, fifty-one years. Maternal grandmother, living, well; age, seventy-four years. Paternal grandmother, living, well; age, seventy-five years.

Diseases Previous.—Measles when two years old; chicken pox when four; whooping cough when ten; malarial fever, eight years.

Physical Examination.—Weight, 160 pounds. Height, 5 feet, 10½ inches. Chest measurement 36 inches expanded, 40 inches insp. Waist measurement, 33 inches; robust, well formed. Heart, lungs, liver, joints, etc., normal. Tenderness and pain over left eye increased on pressure.

Personal Habits.—Lives in a swampy, poorly drained district; is employed as a locomotive fireman. Uses tobacco moderately. Uses alcohol moderately, a glass of beer or whiskey each day, sometimes two; more if on party. Retires at 10 p. m., rises 5 a. m. Sleeps well at night in house not well screened. In the summer time is in the habit of sitting on unscreened porch, and is bothered a great deal by the mosquitoes. Has not been away from home in eight years. Is a big meat eater twice a day, averaging one and a half pounds of meat per day.

Present Illness.—About five weeks ago patient had a heavy cold on his chest, later extending into the nose and pharynx (naso-). This sickness began with a severe rigor, accompanied by pains in the head, particularly at the frontal region. The patient also complained of a bitter taste in the mouth. Had a temperature for the first week, pains in the back and legs, malaise, no appetite, bowels regular, frequent micturition. The pains in his head increased, and Dr. Apgar sent him to the Episcopal Eye, Ear and Throat Hospital. Upon admission he was suffering from pain and tenderness over the left eye and the frontal region of the forehead. Pain was markedly increased upon pressure. No discharge from the nose other than normal. Pain was not increased by the reclining posture.

Clinical Examination.—Urine showed nothing abnormal. Blood showed the presence of a large number of tertian malarial parasites. Scarcely one erythrocyte could be found free from the parasite. The leucocytes were at first decreased, five thousand per cubic millimeter; later there was a leucocytosis, twelve thousand per cubic millimeter.

Treatment—Quinin sulphate, grains five every three hours, was given for the first four days. The malarial parasites showed marked decrease in numbers. The fifth day they showed increasing numbers. The quinin was then increased to grains five every two hours. The parasites again decreased and almost entirely disappeared. The pain over the left eye and frontal region entirely cleared up, and the patient was discharged as cured of the condition for which he came to this hospital.

Diagnosis.—Malaria involving the frontal sinus.

Prognosis.—Favorable.

It is interesting to note that although the patient was infected through a tertian malarial parasite, he had never had a chill, fever, or other evidences of malarial infection. During the two weeks that he was under our observation he ran a normal temperature. I never saw a field in a slide from a malarial patient that showed more parasites in it than did the slide which I examined of this blood. Under appropriate antimalarial treatment the parasites disappeared from the blood. With the disappearance of the malarial infection the frontal sinus pains and tenderness also gradually passed away. The patient was discharged cured at the end of two weeks.

XXXII.

THE INNERVATION OF THE NASAL CHAMBERS.*

BY ALBERT P. BRUBAKER, M. D.,

PHILADELPHIA.

It was a pleasure and an honor to have been invited by the program committee to present to you, though briefly, some of the anatomic and physiologic facts relating to the innervation of the nasal chambers and their associated structures.

The secretomotor and vasomotor nerve mechanisms that excite and regulate the secretory discharge and the vascular supply of this region, and the manner in which they in turn are excited, not only to normal but to abnormal activity, seemed an appropriate subject for presentation and perhaps discussion at a meeting of this character. In the short time at my disposal, only the more essential facts regarding these mechanisms, in so far as they have been described, will be alluded to.

The mucous membrane lining the nasal chambers is thick and spongy and provided with a large number of glands, some of which secrete a fluid rich in mucin, while others secrete a fluid containing albumin, and hence fall into the two categories of mucous and serous glands. The surface of the mucous membrane is covered for the most part with tall columnar cells, between which are to be found mucus or goblet-shaped cells, so called from the appearance they present after the discharge of their contained masses of mucin. The blood vessels distributed to the mucous membrane, and which supply the glands and epithelial cells with the raw materials for the elaboration of their secretions, are mainly branches of the sphenopalatine arteries—subdivisions of the internal maxillary—though branches of the facial are also contributory. Veins which correspond in a general way to the arteries return the venous blood to the general circulation.

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*Read before the American Laryngological, Rhinological and Otological Society, White Sulphur Springs, May 5 and 6, 1916.

In order that the glands may be enabled to elaborate and discharge the necessary amount of secretion, it is essential that the vascular supply be varied from time to time in accordance with the nutritional needs and functional activities of the mucous membrane and its contained glands. The secretion of the glands as well as the blood supply no doubt impart to the nasal chambers that degree of moisture and temperature necessary to prepare the inspired air in both respects for its entrance into the lower respiratory organs.

Though living, and therefore possessing the fundamental property of all living material—viz., irritability—the epithelium lining these glands, as well as the muscle cells in the walls of the arterioles, are lacking in spontaneity of action, and therefore require for the manifestation of their activities the stimulating influence of nerve impulses originating in and transmitted from the central nervous system by specialized groups of nerve fibers. The nerves distributed to these two structures belong to what is termed at the present time the autonomic system, and hence consist of two consecutively arranged neurons.

THE EFFERENT NERVE MECHANISM.

The origin, course and distribution of the secretomotor and vasomotor nerves for the glands and blood vessels of the nasal mucous membrane have been fairly clearly determined in recent years. The results of many lines of investigation are represented in the accompanying diagram.

In a description of this mechanism it will be convenient to consider, first, the postganglionic fibers and their distribution; and second, the preganglionic fibers by which they are associated with the central nervous system. The postganglionic fibers that directly excite to activity the epithelium of the glands and the muscle fibers of the blood vessels have their origin in the nerve cells which constitute the sphenopalatine, or Meckel's ganglion, situated in the sphenomaxillary fossa. From this origin the nerve fibers pass in different directions to become ultimately related, histologically and physiologically, with the epithelium of the glands and blood vessels, not only of the nasal chambers, but of the roof of the mouth, palate and upper portion of the pharynx as well. The stimuli that call

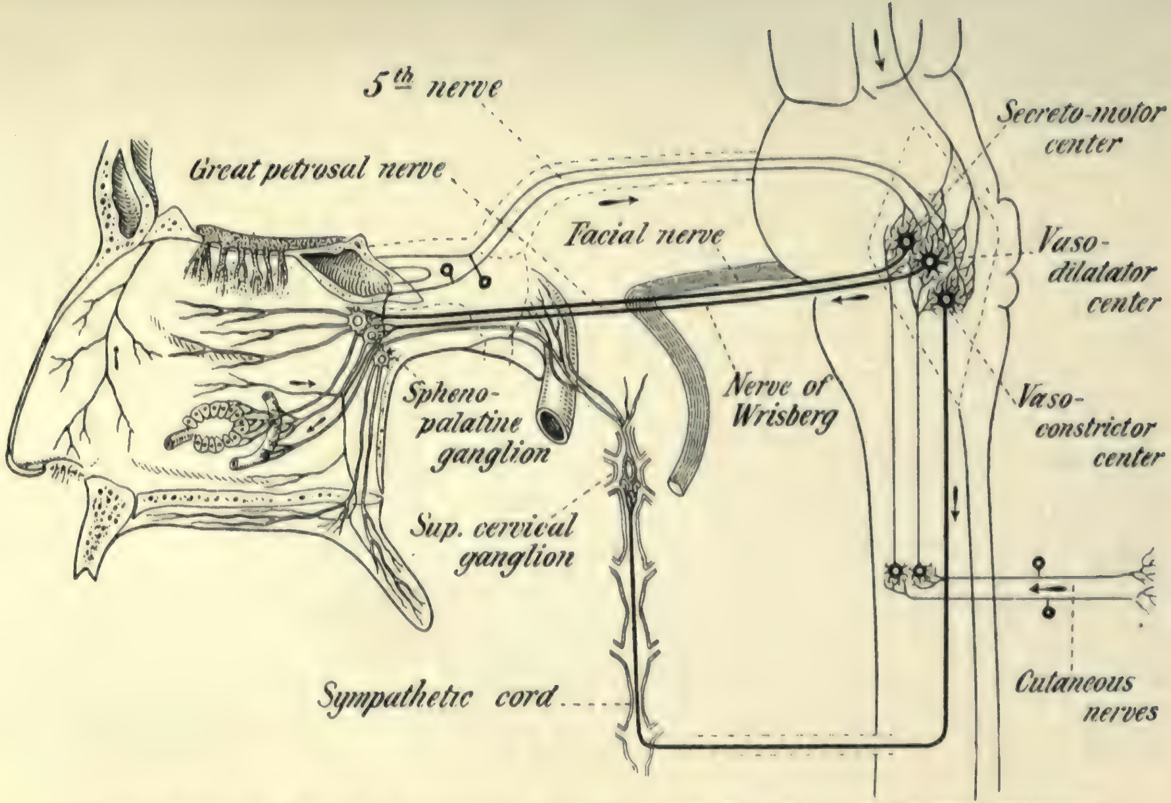

Diagram showing the nerve mechanism concerned in the innervation of the nasal chambers.

forth the activities of these peripheral structures are nerve impulses discharged by the ganglion cells and transmitted to them by the cell processes and their branches. But the ganglion cells, though irritable, are also devoid of spontaneity of action, and require for their excitation the arrival of nerve impulses, discharged by the nerve cells in the central nervous system and transmitted by the preganglionic fibers.

The preganglionic fibers have their origin in nerve cells situated in the gray matter beneath the floor of the fourth ventricle. From this origin the nerve fibers pass forward and emerge from the side of the medulla, in a small bundle, between the facial and acoustic nerves, known as the pars intermedia, or nerve of Wrisberg, of which they constitute a part. Just after these nerves in their forward course enter the internal auditory canal, the nerve of Wrisberg enters the sheath of the facial, having but a short independent course. At the point where the facial nerve turns backward to enter the aqueduct of Fallopius, some of the fibers in the nerve of Wrisberg leave the facial and pass forward to the sphenopalatine ganglion, around the cells of which the terminal branches of the nerves arborize, thus forming histologic and physiologic connections with them. These fibers in their course from the facial to the ganglion are termed in works on anatomy the great petrosal nerve.

The central cells, situated in the ventricular gray, are thus brought into relation directly by their preganglionic fibers, with the ganglion cells, and indirectly, by the postganglionic fibers, with the glands and blood vessels. If, therefore, nerve impulses were to be discharged by the central cells, they would be transmitted by the nerve fibers of the nerve of Wrisberg and their continuations—the great petrosal nerve—to the cells of the sphenopalatine ganglion, which would then become aroused to action; these in turn would then discharge impulses which, owing to the extraordinary branchings of their processes, would be distributed in a spray-like manner to the myriads of epithelial cells lining the glands and to the myriads of muscle cells found in the walls of the arterioles.

That this is the pathway for the transmission of nerve impulses from the central cells to the foregoing tissues is shown by the results of experiment. Thus, if the great petrosal nerve is exposed in one of the higher mammals and divided, and the peripheral end stimulated with induced electric currents, there will arise a dilatation of the blood vessels and a discharge of secretion from the glands of the mucous membrane of the nose and associated structures. Stimulation of the sphenopalatine ganglion or its branches gives rise to a similar series of phenomena. An experiment of this character discloses the fact that this nerve contains both secretomotor and vasodilatator fibers, and the further fact that the central cells from which they arise are divisible into two groups—viz., secretomotor and vasodilatator. There is no evidence that the great petrosal nerve contains any fibers from the facial proper for the innervation of any striated muscle of the palate.

The blood vessels of this region, however, are regulated in the degree of their contraction partly by vasoconstrictor nerves. These nerves also consist of two consecutively arranged neurons and, therefore, belong to the autonomic system. The peripheral one arises in the superior cervical ganglion of the vertebral chain. From this origin the nerve processes—postganglionic fibers—pass upward and assist in the formation of the plexus surrounding the internal carotid artery. At the level at which the great petrosal nerve crosses the artery, some of these fibers enter the sheath of the great petrosal and accompany its branches to their terminations—viz., the walls of the blood vessels and possibly the cells of the glands as well. The preganglionic fibers have their origin in cells forming a part of the general vasoconstrictor center, situated in the gray matter beneath the floor of the fourth ventricle. From this origin the fibers descend the spinal cord as far as the thoracic region, where they pass out from the cord in the ventral roots of the upper thoracic nerves. After a short distance they leave the ventral roots and enter the vertebral chain of ganglia and pass upward to the superior cervical ganglion, around the cells of which their terminal branches arborize. There is thus established a rather circuitous pathway between the vasoconstrictor center and the blood vessels of the nasal mucous membrane and the associated structures. That this is the pathway for the transmission of nerve impulses from the vasoconstrictor center to the blood vessels is shown by experiment. Thus, if the cervical sympathetic cord is divided, not only the blood vessels of the nasal chambers, but of the entire side of the head and face, will dilate and become filled with an increased volume of blood, a condition which will last for some days. If now the peripheral end of the divided cord is stimulated with induced electric currents, the blood vessels at once contract and remain contracted as long as the stimulation is continued.

It would thus appear that the blood supply is under the control of two antagonistic groups of nerves, a dilatator and a constrictor group. The caliber of the blood vessels at any one moment is the resultant of the relative degree of activity of these two groups. According as the one or the other predominates will the caliber of the vessels be increased or decreased, and the blood supply increased or decreased in the same ratio.

THE CENTRAL MECHANISM.

The central mechanism for the innervation of the nasal chambers is thus shown to consist of groups of nerve cells having secretomotor, vasodilatator or inhibitor, and vasoconstrictor or augmentor functions.

The question now arises as to the manner in which these different groups are excited and coordinated in action. Experimental work has demonstrated that as a primary condition for their activity they must possess a certain degree of tonicity or tonus; a degree of excitation due to a reaction between the constituents of the cells and the constituents of the lymph and blood by which they are surrounded. Given this tonus they may be excited to activity or inhibited by:

1. The arrival of nerve impulses transmitted from the mucous surfaces of the nasal chambers by the nasal and other branches of the trigeminal nerve, and from the skin by the cutaneous nerves.
2. The arrival of nerve impulses descending from the cerebrum in consequence of psychic states of an affective or emotional character.

In the former instance the excitation is said to be reflex or peripheral in origin; in the second instance, direct or cerebral in origin. In either instance the resulting change in secretion and vascularity will be proportional to the intensity of the peripheral or the cerebral excitation and the degree of tonus possessed by the central cells themselves. It is evident from a study of pathologic conditions that the tonus of one or more of these centers may be abnormally increased or decreased by changes in their nutrition; changes due to alterations in the composition of the lymph and blood, the result of increased production or diminished elimination of the more or less toxic products of metabolism. It is a widespread observation that the perverted functions of the nasal chambers are closely associated with dietetic or alcoholic excesses, in conjunction with an insufficient amount of physical exercise and fresh air. With the establishment of an impaired tonus of the central mechanism, peripheral causes which at one time would produce slight if any effect, may now give rise to the most pronounced and distressing effects.

THE AFFERENT NERVE MECHANISM.

The afferent portion of this nerve mechanism is to be found in the branches of the trigeminal nerve. The fibers composing the sensor portion of this nerve have their origin in monaxonic cells in the ganglion of Gasser. (See diagram.) The cells of this ganglion give origin to a short process which soon divides into two branches, one of which passes centrally, the other peripherally. The centrally directed branches collectively form the socalled large or sensor root; the peripherally coursing branches collectively constitute the three main divisions of the nerve—viz., the ophthalmic, the superior maxillary, and the inferior maxillary.

The axons of the centrally coursing branches, after entering the pons Varolii, ultimately aborize around the vasoinhibitor, the vasoconstrictor and the secretomotor centers; the peripherally coursing branches pass forward to be distributed to the skin, mucous membrane and other structures of the head and face. By this means the nasal mucous membrane is brought into anatomic and physiologic relation with the foregoing nerve centers. Other of the centrally coursing branches aborize around other groups of nerve cells, the socalled "sensor end nuclei," the axons of which pass upward to and aborize around sensor nerve cells of the cerebral cortex. Stimulation of the first group calls forth reflex phenomena; stimulation of the latter group evokes conscious sensations of a painful character.

The cutaneous nerves associated with this mechanism have a similar origin and distribution.*

REFLEX OR PERIPHERAL EXCITATION OF THE CENTRAL MECHANISM.

The stimulus to the peripheral terminations of the nasal and cutaneous nerves is the variation in the temperature and moisture of the atmospheric air.

If one of the functions of the nasal chambers is the warming and moistening of the inspired air to that degree best adapted for its entrance into the lower respiratory passages, it is apparent that the amount of heat radiated from the blood and the amount of secretion produced by the glands will, or ought to, vary with the variations in the temperature and moisture of the inspired air. Since these latter factors may vary simultaneously in the same or opposite directions, it is apparent that the former factors will. or ought to, vary in accordance with them in the opposite direction.

The phenomena that will be observed are difficult to predict, for the reason that the reactive adaptations of individuals vary with a variety of factors—age, sex, occupation, habits of life, stability of the nerve centers, etc. However, in a general way it may be stated that if a fairly normal individual be subjected to a temperature of sixty degrees Fahrenheit it will be found that the blood vessels of the nasal chambers will possess a certain caliber, the resultant of the cooperative action of the vasodilatator and vasoconstrictor nerve centers. This caliber will permit the passage of a definite volume of blood with a definite velocity in a unit of time, carrying with it a certain volume of heat. By reason of the difference in the temperature between that of the blood (ninety-eight or ninety-nine degrees Fahrenheit) and that of the air passing into and through the nasal chambers—viz., thirty-eight degrees Fahrenheit—heat will at once be radiated into the chambers, and being absorbed by the air will raise its temperature. The degree

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***The ganglia—the sphenopalatine, the otic and the submaxillary—though anatomically associated with the trigeminal nerve, are not integral portions of it; they belong to the autonomic system of nerves and are associated with and constitute a part of the vasomotor and secretory systems of nerves. They possess neither sensor nor motor functions.**

to which the temperature will be raised is difficult to determine, as the absorption of heat will depend very largely on the rapidity and depth of the inspiratory movement, and hence the duration of the stay of the air in the chambers.

If the external temperature should fall to thirty degrees Fahrenheit, the arterioles of the nasal mucous membrane, as well as those of the general cutaneous surface, will contract as a result of the now preponderant action of the vasoconstrictor center, due to the stimulation of the afferent nerve endings by the cold air. This would of course diminish the thickness of the blood stream, a condition which at first glance would diminish heat radiation. But it must not be forgotten that though the thickness of the blood stream is diminished, its speed is increased, so that in a unit of time an equal, or perhaps even a greater, volume may pour through the vessels, carrying with it the same, or perhaps greater, volume of heat. Owing to the now greater difference between the temperature of the blood and that of the inspired air—viz., sixty-eight degrees Fahrenheit—the speed with which the heat is radiated will be increased, and, therefore, the temperature of the air will be corresponding raised. When the temperature of the air returns to sixty degrees Fahrenheit, the former condition of the blood vessels will be reestablished.

Should the external temperature be raised to ninety degrees Fahrenheit, the blood vessels will dilate, the thickness of the blood stream will be increased and its speed diminished. This condition is favorable to heat radiation, but owing to the slight difference between the temperature of the blood and that of the air, the speed with which heat is radiated is correspondingly decreased.

With regard to the secretory mechanism, similar statements may be made. It is well known that a diminishing volume of blood flowing into the capillary vessels surrounding glands decreases the secretion, while an increase in the volume of blood increases the secretion—other things being equal. These two phenomena are always associated in the production of secretions by glands in general. Air that is warm ought, therefore, to increase the vascularity and also the secretion. Under these conditions, however, the additional amount of moisture necessary to approximately saturate the air may render the increase in secretion unobservable. In warm and moist air the secretion increase may be noticeable, because the air, having a high water content, does not take up the water in the secretions, and the excess is drained out of the nostrils.

Air of a low temperature, on the contrary, should theoretically diminish the secretion, by reason of the diminished vascularity and the local action of the cold air on the mucous membrane. Nevertheless, it is within the range of observation and experience that the secretion on a cold day is not infrequently very much increased. As to the manner in which this is accomplished there is much obscurity. A possible explanation for the fact may be found in the effects that follow the action of currents of cold air passing over more or less suddenly exposed regions of the body. It is a familiar observation with many people that the exposure of the cutaneous surface of the head and ankles by the removal of the customary coverings is followed promptly by an increase in the nasal secretion, and possibly a local increase in vascularity. The increase in secretion trickling over the surface of the mucous membrane stimulates the endings of the trigeminal nerve, thus giving rise to the sensation and the reflex act of sneezing. A similar condition is established by the plucking of hairs from the inside of the nostril, or by the inhalation of snuff. It is a possibility, therefore, that though the inhalation of cold air tends to diminish secretion, it at the same time tends to increase secretion reflexly through its action on cutaneous nerves. The effects produced might be regarded as the resultant of the action of two antagonistic forces.

Should the vascular and secretory changes continue, it is possible that under changed conditions of nutrition and lowered vitality the mucous membrane might become the seat of bacterial invasion and activity, with the development of the nasal phenomena characteristic of acute rhinitis. This is especially true in those whose nerve centers have been rendered unstable by dietetic or alcoholic excesses, or by the increased production or defective elimination of metabolic products.

DIRECT OR CEREBRAL EXCITATION OF THE CENTRAL MECHANISM.

That psychic states of an emotional character can influence the secreto- and vasomotor centers in the lower regions of the nerve system, has been observed by everyone from personal experiences and abundantly demonstrated by experiment on mammals by Pavlov, Cannon and others. It is only necessary to recall the effects of the psychic states on the secreto- and vasomotor centers regulating the flow of saliva and gastric juice; in similar centers regulating the secretions and vascular phenomena of the sexual organs. Evidence has been recorded that psychic states of a sexual character have and do cause similar phenomena in the nasal mucous membrane—viz., increased vascularity, increased secretion and erection of its tissues. It has been reported by J. N. McKenzie that a patient of his always had an attack of acute rhinitis after a period of sexual excitement.

These facts, taken collectively, show that the physiologic conditions of the nasal chambers require for their maintenance a complex nerve mechanism; a mechanism which, however, may be easily impaired and rendered unstable by internal causes, whereupon it acts in an abnormal manner, and thus lays the foundation for the development of pathologic states of longer or shorter duration.

XXXIII.

ON THE RELATION OF DISEASES OF THE ACCESSORY SINUSES TO DISEASES OF THE EYE, ESPECIALLY IN CHILDREN, WITH A REPORT OF TWO CASES.

REPORT OF A CASE OF ORBITAL ABSCESS AND ONE OF THROMBOSIS OF THE CAVERNOUS SINUSES.

BY J. H. BRYAN, M. D.,

WASHINGTON, D. C.

The relation of diseases of the accessory sinuses to diseases of the eye is now well established. In the past I have presented several communications on this subject, and reported cases showing the influence of the various affections of the accessory sinuses on the orbit and its contents. Most if not all of these cases occurred in adults. But diseases of the sinuses occurring in children have been only slightly considered, for the reason that these cavities in the very young are supposed to be so small they could not be the seat of an inflammation sufficiently severe to cause any serious disturbance with the eye.

These cavities vary in size in children as in adults, and in many subjects are quite well developed; and when severely inflamed that they have a serious influence on the eye is a well established fact, as will be demonstrated in the report of the following interesting cases:

Case 1. Suppurating Ethmoiditis Causing an Orbital Abscess in a Child Eighteen Months Old.—W. M., aged eighteen months, was admitted to the Episcopal Eye, Ear and Throat Hospital in March, 1914, on the service of Dr. John Burke, with a very marked exophthalmos on the left side.

His mother stated that during the recent epidemic of influenza all the members of her family had been more or less seriously affected by the disease. About a week after this child had been infected, his left eye commenced to protrude from the orbit, and there was a severe swelling of both lids, especially the upper. The family physician applied lead and opium wash to the parts, but no relief being obtained, he brought the child to the hospital.

On admission his temperature was 104°. There was some secretion flowing from the left nostril, and there was a very marked bulging of the left eye downward and outward. The lids and conjunctiva were edematous, and the periauricular glands were enlarged. As the child could not be controlled, it was not possible to make a satisfactory intraocular examination.

The following day I was asked by Dr. Burke to see the case, and we decided it was a case of orbital abscess resulting from an infection through the ethmoid cells. The radical operation was then done, the incision commencing at the junction of the middle and outer third of the supraorbital ridge, and was carried inward and downward along the inner border of the nose below its middle. The periosteum along the inner wall and the corresponding parts of the roof of the orbit was stripped from the bone, and in doing so a large quantity of pus was evacuated. The whole of the inner wall of the orbit was removed back as far as the sphenoid. The ethmoid cells were found to be badly diseased, especially the middle and posterior portion, and from the condition I found, it was apparent the orbital abscess resulted from a direct infection from the middle and posterior ethmoid cells. The amount of pus evacuated was enormous, when we take into consideration the age of the child and the stage of development of these parts at this age. The abscess having been thoroughly evacuated, a strip of iodoform gauze was placed in the orbit back of the eye and brought out through the nose, and a small gauze drain was placed just inside of the inner canthus, and the external wound closed by interrupted sutures.

This child made an uninterrupted and quick recovery, the eye, gradually receding, soon assumed its normal position. This is the first case of such a marked abscess of the orbit resulting from sinus disease in one so young that I have met with, and in the light of my past experience with these conditions, I believe it is most unusual.

Case 2. Chronic Abscess of the Posterior Ethmoid Cells and Sphenoidal Sinus Causing a Marked Exophthalmos in a Child Eleven Years of Age.—Robert C., aged eleven years, negro, was referred to me from Dr. Henning's clinic on the eye service of the Episcopal Eye, Ear and Throat Hospital, November 2, 1915. Giving a history of the present condition having existed about six weeks, on examination the left eye was found bulging to a marked degree downward and outward. The bulging was so great that the lids did not completely cover the ball. There was no secretion from the nose or nasopharynx. Rhinoscopic examination showed only a moderate swelling of the middle turbinal on the left side. Probing in the anterior and middle ethmoid region failed to reveal any caries or necrosis of the bone, and no secretion followed irrigation of the nose.

The following are the findings after an X-ray examination. Stereo plates in the lateral and posterior anterior positions showed the frontals to be normal; the ethmoids clear on both sides; the maxillary sinuses clear on both sides. The left orbital cavity is apparently filled with a dense mass which seems to be confined to the orbit.

In the light of the X-ray findings, and in the absence of any catarrhal condition present, and the negative history of any previous catarrhal state, it was thought we had to do with a growth in the orbit back of the eye. I therefore decided to explore the orbit. An incision along the orbital ridge was made, commencing at its outer third and extending down on the inner side of the nose to its lower third; the orbit was exposed and the periosteum covering its inner wall was elevated. At the junction of the middle and posterior portion there was a decided bulging of the ethmoid toward the orbit. With a probe the cells were perforated, and a large quantity of pus was evacuated. The whole of the inner wall of the orbit, including all the ethmoid cells, were removed as far back as the sphenoid, and in doing so I found a large abscess involving the posterior ethmoid cells and the sphenoidal sinus. The sphenoidal cavity was unusually developed and filled with thick, creamy pus. All diseased bone and purulent secretion having been thoroughly removed, an iodoform gauze packing was placed in the sphenoid and ethmoid regions, one end being brought out through the nose and the external wound closed by interrupted sutures. At the end of the second day the gauze drain was removed and the nose gently irrigated with a saturated solution of boracic acid.

The patient made a quick recovery, the eye gradually receding within the orbit, and at the end of the ninth day he was discharged from the hospital. About ten days later I saw the patient again in the eye clinic, and his eye had assumed its normal position and the vision was perfectly normal.

Photograph No. 1 illustrated fairly well the amount of exophthalmos when I first saw the patient. This case is interesting because of the deceptive character of the X-ray findings, the absence of nasal symptoms, and the unusual size of the sphenoidal sinus in a subject so young.

Case 3. Thrombosis of the Cavernous Sinuses.—This patient, a man of thirty-six years of age, was first seen by Dr. Henning about six o'clock in the evening of November 3, 1915.

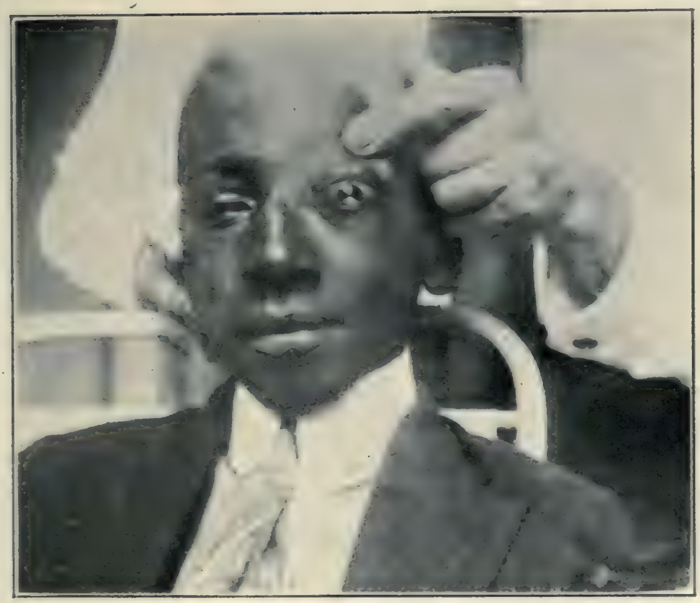
The history Dr. Henning received from the family physician was that the patient in June had an infected hand which was slow in healing, and this was followed by a series of furuncles in both external auditory canals, all of which finally healed under appropriate treatment. During the latter part of October a pustule appeared on the nose, and was opened by the patient. The following day he was seized with severe pain over the left eye, followed soon afterward by exophthalmos on the same side.

When Dr. Henning first saw the patient there was a marked bulging of the left eye, with commencing conjunctival edema and diplopia. The optic nerve head showed a blurring of its edges, and the veins of the retina were a trifle larger than those of the right eye. At this time the right eye was normal in every respect. The temperature was 104°. Examination of the nose showed a moderately swollen middle turbinate on the left side. Irrigation of the nose brought away some thick blood stained mucopus. As the patient was in a very serious condition and growing rapidly worse, he was removed to the Episcopal Eye, Ear and Throat Hospital. At nine o'clock the following morning the exophthalmos was more marked in the left eye, the conjunctiva was so edematous that it overlapped the cornea and protruded between the lids. There was also a commencing exophthalmos on the right side. The temperature was 104.6°. Although quite dull, the patient was able to comprehend the questions put to him, and stated that he was free from pain. At noon I saw the patient in consultation with Dr. Henning, and found the exophthalmos on both sides very marked, conjunctiva protruding between the lids and exuding a bloody secretion. Examination of the nose showed the turbinals on both sides swollen and intensely congested. I probed into both sphenoidal sinuses, and on irrigation brought away a large quantity of thick yellow mucopus.

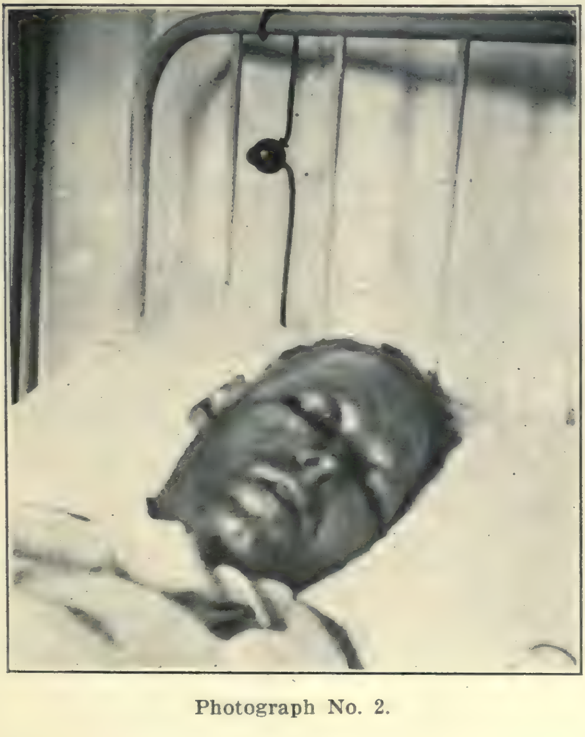
Later in the afternoon, under ether anesthesia, I removed the middle turbinals and opened both sphenoidal sinuses, finding some mucopurulent secretion in both cavities. Examination of the secretion taken from the sphenoidal sinuses showed both streptococci and staphylococci present. A lumbar puncture showed the spinal fluid under great pressure, filled with pus cells, roughly estimated at forty thousand per millimeter. Polymorphonuclear leucocytes predominated. The condition of the patient grew rapidly worse, and he died at 10 p. m.

Postmortem examination, the head only examined, showed all the sinuses contained dark and semifluid blood; small hemorrhages on the right half of the cribriform plate of the ethmoid bone. Vessels of the pia mater generally engorged, the basillar surface of the brain showed fibrin and a purulent exudate under the pia, especially on the insula and adjacent opercula. A microscopic examination showed the vessels of the brain engorged. Beneath the membrane was a thin layer of brown material, intimately adherent to the brain substance beneath. On section the brain surface showed brownish points near the surface; the brown exudate under the membrane showed fibrin, red blood cells and mono- and polymorphonuclear round cells.

Photograph No. 2 shows a typical picture of the conditions found in thrombosis of the cavernous sinuses. This case is not only rare but interesting, and was characterized by a sudden onset in which the initial symptoms were ophthalmic and then meningeal, and death within forty-eight hours. When we take into consideration the free anastomosis of the veins of the nose, pharynx, mouth and face, with the veins of the orbit and the cavernous sinuses, and the absence of valves in the veins, we can appreciate the readiness with which the infection was carried from an insignificant pustule on the face to the cavernous sinuses and then to all the sinuses of the brain.



Photograph No. 1.



Photograph No. 2.

XXXIV.

TWO CASES OF CHRONIC PANSINUITIS ASSOCIATED WITH SYSTEMIC INFECTION.

By George E. Shambaugh, M. D.,

Chicago.

I venture to record the two following cases of pansinuitis, first, because both serve to illustrate the difficulties occasionally encountered in attempting to eradicate the infected foci, and second, because both cases were associated with a serious systemic infection, which presumably took its origin from the infected sinuses.

Case 1.—Miss W., aged thirty-five years, consulted in first in September, 1910, complaining of purulent discharge from both sides of the nose and into postnasal space. She was suffering also from disturbed digestion, which she attributed to the swallowing of the secretion from the nose, especially during sleep. She gave the following history of her nasal symptoms:

The trouble began from an infected upper molar, from which her dentist, in 1895, removed an old filling and killed the nerve because of a severe toothache. In 1897 this tooth was crowned. After this the tooth remained sensitive, and she detected a bad odor from the pus, which continued to escape about the crown.

In 1900 she began to suffer from pain over the maxillary sinus, and noticed for the first time purulent discharge from the nose. In March, 1903, had the crown removed and the diseased root extracted. In April of same year, because of continued pain in upper jaw, was given chloroform and a fragment of necrosed bone removed from the upper jaw. The following June, under chloroform, an opening was made into the antrum from the canine fossa and extended through into the nose. Again in October, same year, a similar operation was undertaken, under chloroform, because of the continuance of the purulent discharge.

After these operations she continued to have a purulent discharge from the nose, and suffered from symptoms of persistent head cold and more or less severe headache, but had nothing more attempted in the way of treatment until 1909, when for the first time she came under the care of a rhinologist. The previous work had been done by a general surgeon. She was again given a general anesthesia, when the antrum was opened from the canine fossa and a satisfactory permanent opening made into the inferior meatus of the nose. Later, under local anesthesia, the middle turbinated bodies, both sides, were removed and the ethmoid cells drained. The patient received a great deal of relief from these later operations, especially from the headache and pain in the region of the frontal sinuses.

No further operation was attempted until she consulted me in September, 1910. An examination at that time discovered creamy pus in both nasal chambers. On the left side pus came from the sphenoid sinus as well as from the frontal sinus and some anterior ethmoid cells. On the right side pus was washed from the antrum and could be traced as coming from the frontal sinus as well as the anterior ethmoid cells. It was also possible to trace pus coming from the right sphenoid sinus. At this time an operation was undertaken, under local anesthesia, on the left side of the nose. The anterior wall of the sphenoid sinus was removed. The remaining anterior ethmoid cells, from which pus was exuding, were removed, and the opening into the frontal sinus was enlarged by means of a frontal sinus rasp.

In February, 1911, patient returned to the city. Discharge from the left side of the nose had become much less, but could still be traced to the frontal sinus and to a narrow canal extending directly backward from a point just below and to the outer side of the floor of the sphenoid sinus. Into this canal a probe could be passed a full centimeter. This infected fistula seemed to be nothing else than the Vidian canal.

She consulted me again in May, 1911. A month previous she had suffered from an attack of articular rheumatism, which began in the ankles and involved later both knees, the hips and elbows. The joints were distinctly swollen. Under local anesthesia an operation on the right side of the nose was undertaken. A large opening was made from the middle meatus into the antrum. A large posterior ethmoid, full of thick pus, was opened. The anterior wall of the suppurating sphenoid sinus was removed.

In October, 1911, patient was seen again. The rheumatism had entirely disappeared. She had been much relieved by the last operation. The headache, which had annoyed her, had entirely disappeared. The discharge from the nose had been greatly reduced. On the right side of the nose the only place from which pus was still exuding was the frontal sinus and from the fistula above. On the left side pus was still coming from the frontal sinus and from the fistula described above. On irrigating the right frontal sinus the water passed through to the left side of the nose, showing a perforation in the frontal septum. The opening into the right frontal sinus was enlarged at this time by means of the frontal sinus rasp.

The condition when patient was last seen in March, 1915, was as follows: A small amount of pus coming from both frontal sinuses and from the fistula on the left side. In spite of the removal of a large part of the inferior turbinated bodies, as well as the whole of the middle turbinates, and the exenteration of the ethmoid labyrinth, both sides, there has been no development of atrophic changes or crust formation other than that forming at the points at which pus was appearing.

Case 2.—Miss M., aged twenty-five years, came under my care in February, 1914, suffering from severe multiple arthritis. She gave the following history: Since childhood she had suffered more or less from nasal trouble. The symptoms were extreme liability to head colds, annoying discharge from the nose, with subsequent irritation of the throat and attacks of acute laryngitis. From 1897 to 1899 she had a number of operations on the sinuses, one of which consisted in the opening of the left maxillary sinus through the socket of a molar tooth. In this opening she wore a tube for about a year, when she removed the tube and allowed the opening to granulate shut. At the time when the antrum was drained an intranasal operation was done to relieve a frontal sinus abscess which was causing excruciating pain in the region of the left eye. For two or three years following this operation had a probe passed from time to time into the frontal sinus.

No further operating was done on the nose until in September, 1912. During this time she continued to be annoyed by the nasal symptoms recorded above. In August, 1910, she had suffered from a severe pain in the left maxillary sinus, which lasted several days only. On January 18th, of the same year, she was seized with a violent attack of acute articular rheumatism. The arthritis still persists and involves every joint in her body.

In October, 1912, work was begun on the nasal sinuses, in the hope of removing the possible focus for the serious systemic infection. The ethmoid cells, left side, were broken down and adhesions were found between the remnants of the middle turbinated body and the septum, the result of some previous operation.

In December, 1913 the left maxillary sinus was opened through the middle meatus and pus washed out. Irrigation of the right antrum at this time disclosed no evidence of pus infection.

I saw the patient first in February, 1914. She was suffering greatly from the arthritis, which involved every joint in the body. She was unable to walk, every joint was swollen and painful. She experienced at times acute exacerbation of the infection, now in one joint, now in another.

The examination of the nose disclosed completely atrophied inferior turbinated bodies. The middle turbinated body, both sides, was absent. There were some crust formations here and there in the region of the ethmoid, more marked on the right side, but no evidence of ozena. A large opening was found into the antrum on the left side. No free pus was found in antrum. The anteroposterior radiogram gave no evidence of a frontal sinus either side. It was with difficulty that a small probe could be passed into the opening of a small frontal sinus on the left side. The withdrawal of the probe was followed by a drop of pus. From the right maxillary sinus a quantity of thick mucopurulent secretion was washed out. A large opening was made at this time from the middle meatus into the right antrum. The patient has since been able to irrigate both maxillary sinuses herself, and both have remained practically free from secretion, except for a short period following acute head colds.

The left frontal sinus has been repeatedly irrigated and, as a rule, a small amount of granular pus washed out.

In January, 1915, by exploring carefully with a probe the locality in the right nares where crusts were seen to form, a large posterior ethmoid cell, filled with thick creamy pus, was discovered and drained. At the same time the opening of the right sphenoid sinus was discovered in the same way and the anterior wall removed. The sinus was filled with the same kind of pus. Two weeks later, by the same method of exploring with a probe the region in the front of the nose where crusts were forming, an ethmoid cell was discovered, also filled with thick pus. I was not able to discover an opening to a frontal sinus on the right side.

In February, 1915, two small ethmoid cells on the left side were found secreting pus and were drained. The crust formation on both sides of nose much improved. Present condition of patient, as far as the nose is concerned, seems to be quite satisfactory. There is no crusting, in spite of the entire disappearance of the inferior and middle turbinated bodies and the exenteration of the ethmoid. The patient feels more comfortable when she irrigates the nose once each day, but washes out very little secretion and no crusts. A careful examination fails to discover any distinct evidence of pus in any of the sinuses. The general condition of the patient is not at all good. She is free from acute exacerbations of the joint infection and the associated rise in temperature, but the inflammatory changes in the joints are apparently undergoing secondary fibrous changes, which is resulting in greater and greater rigidity. These changes are producing, probably through pressure, more or less continuous pain in all the joints in the body.

Chronic infection in the nasal accessory sinuses is rarely limited to a single sinus. The process not infrequently begins as an infection in a single sinus, as when the antrum is infected from a diseased tooth, but, as a rule, other sinuses become sooner or later involved. This is especially true of those accessory sinuses which open into the middle meatus. The infundibulum often forms a more or less closed trough, which opens at its anterior end into the frontal sinus, and at its posterior end into the maxillary sinus. This trough receives drainage as well from the anterior ethmoid cells. By means of the infundibulum, pus secreted from either the frontal sinus or the anterior ethmoid cells drains directly into the maxillary sinus when the head is in the upright position. On the other hand, when reclining, pus from the antrum is conducted by the infundibulum directly into the frontal sinus. I have occasionally encountered chronic infection limited to the sphenoid sinus, but it is much more common to find combined empyema of all the accessory sinuses on one side.

The method of choice in dealing with chronic accessory sinus infection, whether the process involves one or several sinuses, is by carefully conducted intranasal operations performed under the use of a local anesthesia. The operations conducted in this way are safer, and are, as a rule, as efficient as under a general anesthesia. The one sinus most difficult to approach by the intranasal route is the frontal, and here the disfiguring scar always left after an external operation prohibits this method of operation except for the relief of an impending intracranial complication and after all intranasal methods have failed to relieve the pressure and pain from the confined pus.

XXXV.

A REPORT OF TWO SEVERE CASES OF ULCEROMEMBRANOUS ANGINA.*

By Ira Frank, M. D.,

Chicago.

There occurred recently in my service two unusual and severe cases of ulceromembranous angina, which were of interest not only because of their severity, but also because of the widely differing clinical forms which they assumed. The contrast of these two cases, which I was fortunate to be able to study simultaneously, was so marked—considering that they both represented the same disease, with presumably identical etiologic factors—that I feel that a brief report may be of interest to you. Moreover, a rather diligent search through the literature has shown a marked dearth in the number of serious and fatal cases reported, and has further revealed the existence of debatable points in etiology and treatment. It is, therefore, that I present these two cases in the hope of provoking a general discussion and a recital of personal experiences.

Case 1.—L. S., male, aged forty-eight years, a peddler, was admitted to my service on June 4th. He gave a meager history, which indicated that five weeks previously he had suffered with a peritonsillar abscess which when opened had discharged a large quantity of pus. Some time after admission he was able to tell us that following this evacuation the symptoms continued without remission, though there was no further formation of pus, and his condition grew more serious day by day. He presented himself at the hospital in a pitiful condition, unable to swallow or to open his mouth beyond a one-half inch separation of the incisors. He was weak and emaciated, having lost approximately twenty-five pounds since the onset of his illness.

General physical examination negative. Examination of the

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mouth revealed an area of deep necrosis on the right side of the soft palate, oval in outline, extending from and involving the tip of the upper pole of the tonsil, anteriorly to the hard palate, and mesially to the midline. The area involved was composed of a greenish gray, foul-smelling slough, which was easily removed with very little bleeding, and exposed an ulcer one to one and one-fourth inches in diameter. The base was not raised and only slightly injected. The edges were irregular and vertical, with a punched out appearance. The floor was incompletely formed; a green seminecrotic slough, with a perforation through the soft palate large enough to permit a fair view of the superior portion of the nasopharynx.

The clinical diagnosis, from the picture presented and the history obtainable on admission, was not easy—lying between carcinoma, tertiary syphilis and Vincent's angina. Direct smears from the depths of the ulcer showed pus, fibrin, pneumococci, a fusiform bacillus and spirillæ, with the latter organisms predominating.

The bacilli were five to ten microns long, with tapering ends and homogeneous body. The spirillæ averaged ten microns in length, stained deeply with anilin gentian violet stain. The spirilla contained an irregular number of spirals. Aerobic cultures negative, pneumococcus and staphylococcus being present. Blood findings were normal. Wassermann reaction, taken twice, was negative.

A piece of tissue from the floor of the ulcer was submitted to the laboratory for microscopic examination, and the following report was returned: "No malignancy evident. A suppurative process with marked necrosing tendency. Pus cells numerous, with a small round celled infiltration. Infective cellulitis with gangrene."

The patient was given a frequent gargle of peroxid, with daily deep injections of sodium cacodylate in gradually increasing quantities. It was necessary to administer opiates a number of times after admission, on account of his severe pain.

On the third day of his stay at the hospital the first signs of improvement were noted. This became gradually more pronounced, interrupted only by symptomatic remissions which were controlled by sedatives. There was no evidence of further spreading of the process, and by the fifth day there appeared definite signs of healing in the ulcer. The edges assumed a more normal appearance, and removal of the reforming necrotic center revealed a rapidly healing base.

On the eleventh day the patient was free from pain and the cavity was markedly diminished in size, with cicatrizing edges. The course of the entire process was ten weeks, six of which were spent in the hospital. The patient was discharged in excellent condition. The soft palate was distorted by a firm scar, the center of which contained a narrow fistula, admitting a small silver probe, leading up into the posterior nares.

Case 2.—E. S., female, aged twenty-two years, was admitted to the hospital on June 21st. For one week previous she had complained of severe sore throat, with almost total dysphagia and the usual constitutional symptoms accompanying a severe throat infection. Cultures taken during this period were negative, and smears from the throat showed large numbers of both fusiform bacilli and spirillæ. She was given peroxid gargle without any alleviation of her symptoms, and because of her practically complete inability to take nourishment, she was placed under hospital care.

General examination was negative. On inspection the right tonsil was found covered with a greenish gray membrane which extended beyond the tonsil anteriorly and posteriorly, hanging in small islands on the pharyngeal wall. The membrane was adherent and tough, and rested on an angry red base. It was removed with difficulty. The uvula, soft palate and pharynx were edematous. A large piece of thickened membrane was removed and sent to the laboratory for examination. The report showed necrotic tissue with hyalin degeneration, considerable round celled infiltration and occasional patches of pharyngeal mucosa, surrounded by coagulated débris. Laryngeal examination revealed marked redness of the larynx, especially of the epiglottis, and no edema. Nasal examination negative. Schick reaction negative. Urinalysis negative. White blood cells, 24,000. Differential: Seventy-six per cent polynuclears, two eosinophiles, eighteen mononuclears. Temperature mean, 102°. A gargle of peroxid was ordered for frequent use, and also twice daily deep injections of sodium cacodylate in three-fourths grain doses.

There was no appreciable symptomatic improvement following the introduction of this treatment, though examination showed a less acute process on both tonsil and pharynx, and the membrane covered smaller areas than when first seen. The patient complained more bitterly of pain. She became hoarse, was unable to talk above a whisper, and no manner of coaxing or scolding would induce her to swallow more than a few drops of milk or ice water at long intervals. Deglutition caused severe attacks of coughing which were pitiful to witness. She complained of an intense substernal burning pain, lasting fifteen to twenty minutes after swallowing minute quantities of water. An attempted laryngeal examination on the second day showed the presence of patches of membrane and edema of the epiglottis. Examination of the cords was unreliable, because of strenuous objection by the patient.

The situation becoming more and more serious, an effort was made to stay the process by increasing the cacodylate dose to two grains, the intense dysphagia and substernal burning with the increasing signs of laryngeal involvement indicating an extension into the esophagus and larynx.

The urine was at all times scanty, and on the third day showed very positive albumin, hyalin and granular casts. Fluid intake less than eight ounces, practically all of which was water.

Late in the second hospital day the patient exhibited signs of collapse. The cause being attributed to lack of nouishment, and because she was unable to retain nutriment per rectum, five per cent glucose solution was administered intravenously. On the third day the picture remained unchanged, slight improvement noted in the pharyngeal lesions. The urine was scanty and test showed a heavy deposit of albumin. Signs of gradual collapse continued. The patient became irrational and refused to swallow any nourishment. The fourth day was marked by no change in the condition. Toward evening the patient suddenly had a severe retching spell, lasting nearly five minutes, at the end of which she vomited a piece of membrane about four and one-half inches long and one inch wide, thick, with straight smooth sides and irregular ends—evidently a large portion of a cast of the esophagus. Toward morning of the fifth day the patient coughed and suddenly became dyspneic and cyanotic. Emergency tracheotomy was about to be performed when a piece of membrane was forcibly expelled, relieving the symptoms as suddenly as they had appeared. This specimen was three inches long and about half an inch wide—no doubt, a good portion of the tracheal and laryngeal membrane.

There was no appreciable alleviation of symptoms following these incidents. For four days following she had an average intake of five to seven ounces of fluids, and a total twenty-four hour excretion of eight to ten ounces. The pulse became irregular and slow, and examination showed a toxic myocarditis present. Mental symptoms became prominent; the patient satisfied herself that she was going to die, and it was impossible to obtain her cooperation in any therapeutic measure. She exhibited a taste perversion which was marked. Water caused her intense agony when swallowed, but she was certain that sarsaparilla "pop" would go down easily. We procured some, which she drank with apparently not much difficulty. The idea of impending death was so fixed that she prevailed on her people to take her home to die. Exitus occurred on the second day after leaving the hospital.

These cases, both clinically very malignant, present markedly differing types. The first was ulcerative and necrotic, a deep slowly extending destructive process, with symptoms relating only to movement of the affected parts. There were no systemic effects, not even an elevation in temperature, and the lesion responded readily to treatment. The second case was strikingly different. The process was primarily membranous, with only a slight tendency to superficial ulceration. The membrane spread rapidly—evidence of its migration in both the trachea and esophagus being given above. Symptomatically the case was pronounced toxic in features, fever varying from 99° to 103°, nephritis and myocarditis being complications.

What influence, working on the same etiologic factors, produces this widely differing clinical picture? It has been suggested that the course of the disease is modified by the relative numbers of spirillæ and fusiform bacilli present—that predominance of one type of organism or the other produces the varying clinical picture. Such a condition we were unable to prove in these two cases.

It may be suggested that the slow ulcerative type is a chronic infection, whereas the membranous type is an acute or fulminating example of the same disease. I am inclined to hold the latter view, accounting for the ulcerative form as a low grade infection, due either to attenuated organisms or an immunizing protective effort of the body. Examination of the microscopic specimens in the first case certainly show such a condition—the lesion was well limited by a leucocytic wall.

The fulminating character of the second case may well be ascribed to infection with virulent organisms in a host physically unfit to offer resistance and combat the process. As to a comparison of the clinical course, little explanation is necessary. The first case produced no more systemic results than would a walled off abscess; the second patient showed systemic effect due to (1) the virulent infection, (2) absorption in the large area involved, and (3) consequent secondary infection.

XXXVI.

EAR AFFECTIONS AND DIABETES.*

By Chas. Zimmermann, M. D.,

Milwaukee.

Although we do not know of any ear disease peculiar to diabetes, there is no doubt that diabetes has an unfavorable influence on the course and healing of ear affections, imparting to them a certain anatomic and clinical character, and, especially with regard to operative measures, its presence demands the most serious consideration. On the other hand, ear diseases may elicit the chief symptom of diabetes, viz., glycosuria. (Grunert,[1] Frey,[2] Furet.[3])

To begin with affections of the external ear, obstinate eczema of the auricle and the external meatus with annoying pruritus has been observed. Moos reports such an experience on himself. This is in accordance with the skin affections on other parts of the body in diabetes. More severe are furuncles of the external meatus, which often heal very slowly (several weeks—Srebrny[4]), even after incision, and, according to Wolf, have a great tendency to relapses or may lead to coma. Kipp reported a case in a child, affected with diabetes, who in spite of extensive incisions died after twelve hours in coma. In a diabetic patient of Eulenstein,[5] a woman, aged fixty-six years, a severe phlegmon developed after a circumscribed external otitis. Large incisions were made to the bone, which was denuded of periosteum, with free drainage, but the patient died seven days later in coma. Grunert[6] found in Schwartze's clinic in furunculosis diabetes not more frequently than after the indiscriminate urine examinations of all stationary ear

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***Read before the second annual state meeting of the Wisconsin Eye, Ear, Nose and Throat Specialists, under the auspices of the Milwaukee Oto-Ophthalmic Society, October 8, 1915.**

patients, and does not consider diabetes as a predisposing element for the constant recurrences.

Miot[7] states that diabetics suffer at times from ear troubles of varying intensity and duration, which are caused by swelling of the eustachian tube. Some of the cases are curable, in the others the tubes are permanently obstructed and impermeable for air or bougies. (The constant current allays the congestion of the mucous membrane, and renders possible the introduction of bougies and the application of chemical and galvanic cauteries.)

Valentin[8] described that in diabetes oidium albicans may enter the eustachian tube from the nasopharynx to the tympanic cavity and produce otitis media with perforation of the membrana tympani.

Of all ear affections in diabetes, purulent otitis media has been observed most frequently. According to Koerner's statistics,[9] and a critical analysis of forty-six cases by Eulenstein, partly observed by himself, partly from literature,[10] diabetic persons have a greater tendency to suppurations of the ear and temporal bone than nondiabetics, and acute otitis media in diabetics leads much more frequently to diseases of the bone, to extensive caries and necrosis, than in nondiabetics.

Grunert[11] had the same impression from his experiences in Schwartze's clinic. Although the formerly held opinion of a specific diabetic otitis has been refuted as erroneous, it is certain that the greater vulnerability of the tissues of the body in diabetes predisposes to the occurrence of acute otitis media and retards the healing process, so that the affection is apt to become chronic.

This predisposition in diabetes is given by the presence of sugar in the blood and tissues, furnishing a very good culture soil for microbes. It is well known from experimental bacteriology that animals that are ordinarily proof against certain microbes can be successfully inoculated with these after grape sugar has been introduced into the body and an artificial diabetes produced. Thus Bujwid created local gangrene of the skin of animals by injecting very slight, in themselves harmless, quantities of staphylococcus aureus under the skin and simultaneously grape sugar into the auricular vein. Likewise, von Mering and Minkowski[12] observed that their glycemic dogs, after extirpation of the pancreas, inclined to suppurative processes and with one exception never showed aseptic healing. Also Ernst[13] proved clinically and experimentally that diabetes creates a disposition and favorable soil for the growth of the most different lower fungi. Hence, one must assume that the cells of the tissues must be changed by the sugar, so that they do not offer sufficient resistance to morbid agents.

This is further due to lack of nutrition from early arteriosclerosis in diabetes, as first pointed out by Israel. Also Partsch[14] found in diabetes endarteritis of the smaller arteries, with diminution of the lumen, calcification and even ossification of the media, which play an important rôle in the etiology of spontaneous diabetic gangrene. According to the histologic investigations of Hildebrandt,[15] diabetes leads early to degeneration of the contractile vascular walls, secondarily to a proliferation of connective tissue. Clinically he proved this by examination with Roentgen rays. His skiagraphs showed intense sclerosis of the arteries and veins. Intestinal hemorrhages, extravasation in skin and mucous membranes—e. g., of the mouth—epistaxis, hemorrhagic otitis media, subcortical hemorrhages, described by a number of authors (e. g., Sendziak[16]), find their explanation in these vascular alterations. Hence Garel and Armand,[17] who reported thirteen cases of epistaxis in diabetes, recommend the examination of the urine in frequent relapses of epistaxis.

The occasional serious character of acute otitis media in diabetes is illustrated by a case of Chorazycki.[18] The patient, aged twenty-three years, felt greatly relieved after paracentesis. The next day his condition changed for the worse; he had a low pulse and died on the second day. A case of Heiman,[19] who came to him in the fourth week of an acute otitis media, had temperature of 39.5° C. and three and one-half per cent sugar in the urine. The suppuration ceased after a week, and the perforation of the membrana tympani closed. Two weeks later he became unconscious and died under the symptoms of meningitis.

The following cases of acute otitis media and acute exacerbations of chronic purulent otitis media in diabetes took a favorable course, not much deviating from the ordinary form:

Case 1.—March 12, 1912, I saw in consultation a feeble man, aged sixty-five years, who had been affected with diabetes for years, on account of intense pain in his left ear for two days from otitis media. There was a slight discharge, and the meatus was very much swollen A large paracentesis relieved him at once, and he made a good recovery.

Case 2.—On December 31, 1892, I was called to a man, aged fifty years, diabetic for years. He was in bed after an operation for empyema of the pleural cavity, and now complained of pain and discharge in left ear. Large perforation in anterior quadrant, and very offensive secretion. Under treatment the discharge stopped February 17, 1893. March 15, 1899, ear ran again after a severe cold; again on June 6, 1899, which soon healed. The patient died a few years later. .

Case 3.—H. B., aged fifty-two years, diabetic, who had since childhood a discharge from his right ear, came on July 28, 1914, on account of pain and recent profuse suppuration of this ear. There was a small perforation in anterior portion of the membrana tympani, the posterior portion was bulged forward and the mastoid process was very tender; temperature, 99°. Apparently the posterior portion of the tympanic cavity was shut off by adhesions. I therefore made a large curved incision in the posterior portion of the membrana tympani and up to the perforation, which at once relieved him. The pain in the mastoid subsided, and after a few weeks the discharge from the ear ceased.

According to Koerner, adults, especially men, suffering from diabetes, do not easily recover from an acute otitis media without severe, very rapidly spreading mastoiditis. It is characterized by extensive central destruction of bone in the mastoid process, before signs, tenderness to the touch or swelling, show on the outer surface. Apparently it commences by hematogenous infection of the bone, extends early to the pneumatic cavities, and discharges its pus on the preformed path through the tympanic cavity, and at first has the aspect of mere suppuration of the middle ear. The mastoid may be converted into a large cavity, filled with granulations and pus, covered by a very thin corticalis, and the operation reveals the inner table of the sigmoid sulcus or the middle cranial fossa destroyed to a large extent. In these cases a valuable diagnostic means of recognizing the serious condition is percussion of the bone, originally devised by Lücke and further developed by Koerner. From the experience of Lucae's clinic, Grossmann[20] reports that out of the cases of acute otitis media which came to operation 72.7 per cent occurred in diabetic, 56.3 per cent in nondiabetic, patients, due to the alteration of the tissues by diabetes, as set forth above.

All microorganisms that cause otitis media were found in diabetic mastoiditis, viz., streptococcus, staphylococcus albus, and diplococcus, which seems to have a greater tendency to spread beyond its original field of infection, and mixed infections. As further complications have been observed: Necrosis with formation of sequestra (Massier,[21] Koenigsbauer[22]), propagation of the suppuration to the dura, thrombosis of the sinus (Leidler[23]), brain abscess (Heimann[24]), periarticular abscess of the maxillary joint (Ephraim[25]), paralyses of the abducens (Luc[26]) and the facial nerves (Castex[27]).

Affections of the inner ear in diabetes have been considered rare. In 1892, Moos[28] wrote: "Little is known about diabetic affections of the labyrinth." Sendziak[29] considers affections of the inner ear in diabetes much rarer than those of the middle ear. They occur in form of vertigo, tinnitus, and progressive hardness of hearing. Heiman, who is of the same opinion, observed a woman, aged seventy-two years, who became deaf in both ears from labyrinthine disease and diabetes. The hearing returned after two weeks' use of Carlsbader water.

Very interesting is the following case of Lermoyez,[30] diagnosed as anemia of the labyrinth: A man, aged forty-three years, diabetic, who had syphilis and formerly had been addicted to alcohol, suffered, after a cure of abstinence, from tinnitus, vertigo, and hardness of hearing for three and one-half years. The reddened promontorium shone through the retracted membrana tympani. Under strict diet the condition deteriorated. Pilocarpin injections were without result. After meals the patient felt heavy in the forehead, but could hear better, and the noises were diminished for from four to five hours. From this Lermoyez inferred an anemia of the labyrinth, which was favorably influenced by the congestion of the brain occurring during digestion in dyspeptics. The improvement of hearing by inhalation of amyl nitrite supported this supposition.

In some cases the labyrinthine symptoms probably may be due to neuritis of the auditory nerve, an assumption which is admissible if we remember the not rare occurrence of neuritis and atrophy of other nerves in diabetes—e. g., the optic nerve, the sciatic, anesthesiæ and paresthesiæ and neuralgiæ—e. g., of the mastoid (Grunert).[31] This has been corroborated by the anatomic investigations of Steinbrügge, who found in a diabetic individual a hemorrhagic otitis media with microscopically visible extravasations of blood in the labyrinth, at the periosteum of the internal meatus, between the fibers of the auditory nerve and its branches, in the spiral ligament, the endosteum of the vestibulum, and on the neuroepithelium of the ampullæ. According to the conclusions of Friedrich, based on the large material of the ear clinics of Halle and Kiel, there is about one case of suppuration of the labyrinth to every one hundred cases of suppuration of the middle ear. Bezold,[32] however, regards of chronic general diseases diabetes, aside of tuberculosis and lues, as the chief etiologic factor in the proneness of suppuration of the middle ear to advance to the spaces of the labyrinth. This is not surprising, if we consider the tendency of otitis media in diabetes to caries and necrosis mentioned above. The following case may illustrate this:

Case 4.—A sickly looking man, aged thirty years, came to me on April 1, 1898, on account of his hearing rapidly failing within the last three weeks. Right ear was totally deaf. Left ear almost deaf, showed chronic purulent otitis media with very offensive discharge. A broad polypus at the medial wall was removed with curette, and the root cauterized with nitrate of silver. The bone was carious in a large extent. The next day his deafness had increased, and there was some dizziness, but no pain or headache. The parents had noticed the deafness in his third year. Ten years ago he acquired syphilis with secondary symptoms on skin, mouth, tongue and throat.

On April 6th the patient stated that he had diabetes, and showed me a certificate of an examination of the urine made on January 15th by a chemist in New York, who found seven per cent sugar. Now the amount was the same. The right eye was blind, due to traumatic cataract from an injury twenty years ago. Vision in left eye, 15/70; myopic astigmatism; with his glasses, 15/50. The sight of this eye was failing for about a year. The condition remained the same the following few days. On April 7th he complained of pain in mastoid, without visible signs. The patient then went to New York, and, as I heard later, died on April 20th, but I could not find out under what symptoms.

The occurrence of labyrinthine symptoms in this case after the operation, and in another one after the removal of a large polypus of the inner wall of the tympanic cavity with the snare, without injuring the bone, seems to me due to the fact that the fistula in the carious bone was stopped by the polypus, so that lymph could not, or only very scantily, escape. After removal of the polypus the stoppage apparently ceased and the labyrinthine symptoms set in.

I found a similar explanation in a case of acute purulent otitis media in a diabetic reported by Ephraim,[33] in whom the mastoid operation disclosed a fine fistula in the medial wall of the antrum, leading to a fistula in the horizontal semicircular canal, from which lymph oozed slowly. There were no labyrinthine symptoms. Ephraim attributes this, as also Lucae pointed out, to the slight and very gradual efflux of lymph fluid, as it is very frequent in carious, in comparison to traumatic, defects.

With regard to the prognosis of the operation of mastoiditis in diabetic persons, a perusal of the otologic literature shows discrepant views. While some, mostly older authors, consider it contraindicated, others advise not to operate early, the majority, especially recent authors, regard the operation as a vital indication. E. g., in 1895 Buck stated, before the American Otological Society, that the ten cases of Kuhn and Koerner were all the published instances of mastoid disease occurring in diabetic persons which he had been able to discover. He reported four of his own, of which two died after the operation. He considers it imperative that mastoid operations upon diabetic persons shall be performed at a comparatively early stage of the disease in the temporal bone, that is, before the lateral sinus or the dura mater has become seriously involved.

Since then the subject has been repeatedly discussed, e. g., before the Otologic Section of the New York Academy of Medicine, the Otological Society of New York, the Otological Society of Paris, etc. To mention only a few: Richards[34] operated on nine cases, of which six died of coma; McKernon,[35] thirteen, all in chloroform narcosis; four died. Dench, three; one died. Koerner[36] discussed the operative results on thirty-three cases, discriminating between mild, medium, and severe, of diabetes. None of the light form died; the wounds healed more slowly than in nondiabetic patients, but none developed the severe type after the operation. Also all of the medium kind were cured. Out of the nine individuals suffering from the severe form of diabetes, three died from coma; one from general weakness. All were operated upon in chloroform narcosis. In the mild forms Koerner considers the prognosis good, and the operation also permissible in the severe forms, if there is a vital indication. If, however, the urine shows ferric chlorid reaction, the surgeon must not conceal the possibility that the necessary operation may bring on the threatening coma.

For illustration, I may here report the following case:

Case 5.—Mrs. F. D., aged forty-eight years, came to me in January, 1909, with the history that her left ear had been discharging for about three months. For the last few nights she could not sleep on account of severe pain in ear and head. The left side of the head, temple and mastoid region were intensely swollen, and the auricle was standing off. The walls of the left external meatus were very much swollen, making the otoscopic examination difficult; pus was oozing out, and in the depth there was lively pulsation. As the patient suffered from intense pain, she was at once sent to the hospital, where an hour later I opened the mastoid. After the incision pus oozed. The external surface of the mastoid was discolored to a large extent. After its removal an immense cavity was laid open, filled with granulations. At 4 p. m., temperature, 99°; pulse, 130. The next morning, temperature, 98°; pulse still 130. The patient felt greatly relieved and no pain whatever, but from her laborious breathing I suspected imminent diabetic coma, and asked for the report of the urinary examination. This had been omitted in the rush of getting her ready for immediate operation. The test now revealed abundant sugar. Upon inquiry, her physician, who had assisted at the operation, but had not mentioned anything of diabetes, told me now that she had it for three years. Bicarbonate of soda, 2.00, every hour, milk and oatmeal, and injections of salt solution were ordered. On the second morning the wound was dressed and found in excellent condition. The patient was conscious and felt comfortable, but the breathing was more laborious. At 6 p. m. the coma was fully developed, the patient was unconscious, and died at 8 p. m.

If I had known beforehand that the patient had diabetes, I would have advised the operation, which was absolutely necessary, but would have guarded the prognosis and mentioned to the relatives the possibility of coma.

If we consult the modern opinions of internists and surgeons as to surgical procedures in diabetes, I cannot do better than to quote from the views, based on unusually large experience, of Kraus, Naunyn, von Noorden, Minkowski, and the surgeons Payr and Karewski, propounded in the common meeting of the Societies of Surgery and Internal Medicine at Berlin.[37] Every patient submitting to surgical treatment must be carefully examined for diabetes. The unfavorable influence of surgical interference may be caused by the psychic shock and the effects of the traumatism on the nervous system, the deleterious influence of the narcotics on the organs participating in the metabolism of the carbohydrates and the acidosis bodies, loss of blood, disturbances of taking nourishment, respiration and circulation, etc. The consequences may be increase of the intensity of diabetes, as shown by the increase of glycosuria observed after mastoid operations, acidosis, acceleration of general weakness and heart failure. The diabetic patient in need of surgical aid is threatened by two dangers: augmented violence of all kinds of septic processes, and the greater predisposition to coma. The first danger can be met by strict asepsis, under which wound healing takes place in diabetes of any form without disturbances, under avoidance of all toxic antiseptics, by adopting the most simple and rapid method of operation, guarding against injuring the tissues by infiltration, anesthesia, caustics, etc. Infectious processes, however, take a malignant course and increase the dangers of metabolic disease.

Every form of diabetes requires careful consideration, according to the strictest principles of internal medicine, before, during, and after operation. These consist in previous dietetic treatment, aiming at reducing the hyperglycemia, prevention of acidosis, preservation of the strength of the patient, and in proper cases abundant ingestion of alkalies for neutralization of the abnormal acids. Here the starvation treatment, devised by Frederick Allen, must be considered, which is simple, safe, and very efficacious in rendering and keeping a patient sugar-free in a much shorter time than it was possible by the old methods.

Coma is the most dreaded enemy of our therapeutic efforts. Neither the light form of diabetes, nor its successful preparatory treatment, insure against coma after operations, which under all circumstances have the significance of a damaging factor, be they ever so slight, as I have observed in the following case:

Case 6.—G. S., a stout, healthy looking girl, aged nineteen years, came to me on August 17, 1903, being blind in both eyes. The strong aceton smell of her breath at once suggested the diagnosis of diabetes, which was verified by the large amount of sugar in her urine. She weighed 117½ pounds, suffered from thirst and voracious appetite. Both eyes showed total diabetic cataract. To prepare her for operation, antidiabetic diet was ordered. When she returned on September 7th, the quantity of urine, which had a strong smell of aceton, in twenty-four hours was still ten quarts. On September 8th a discission of the anterior capsule of right lens was made in cocain anesthesia. The eye bore this very well, but on September 12th, a. m., she had temperature of 101.6°, dyspnea, pulse 96, and headache, foreboding diabetic coma. Afternoon temperature, 102°, pulse 120. The ferric chlorid test revealed diacetic acid in the urine. Large doses of bicarbonate of soda were given every hour. September 13th, the patient was still conscious, but collapsed and cyanotic, pulse more rapid, less tension. The parents, who were sent for, took her home, where she died on September 14th.

Hirschberg had a similar experience.

Postoperative coma may set in after any kind of narcosis and anesthesia, but especially after chloroform. Chloroform is apt to produce greater acetonuria than ether. Hence Kausch[38] principally denounces chloroform, and considers ether as the narcotic for diabetics. The inhalation narcosis ought to be instituted in the early morning, in order not to prolong unnecessarily the time of the longest physiologic abstinence from food.

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XXXVII.

ESOPHAGOTRACHEAL FISTULA.

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Fistulous communication between the esophagus and air passages was recognized many years ago. The literature abounds in numerous cases, the majority of which deal with the congenital variety, to which the greatest attention is paid.

In 1880 Morell Mackenzie[1] collected all the reports of malformations of the esophagus to date, and found thirty-seven examples in which there was a communication between the esophagus and the trachea or one of the bronchi. The similarity of his observations with those of more recent times stamps this condition as one concerning which very little doubt as to diagnosis or prognosis obtains. The symptoms were those that were to be expected when a communication between the food passage and the air passage exists—namely, inability to swallow, violent coughing and choking, the ejection of swallowed liquids, rapid loss of strength and early death from inanition or pneumonia. Other cases of congenital malformation with fistula have been reported from time to time, the total number collated by Zeit[2] in 1912, making fifty-five. A most interesting study of the etiology of these congenital defects may be found in Virchow's Archiv.[3]

Recently, Losee,[4] Finkelstone and Ellis,[5] Schmidgall,[6] and Robertson[7] have added several cases. But while a study of these congenital cases is of great interest and of more or less importance in a consideration of acquired lesions of this description, it is to the latter that I wish particularly to call your attention.

Mosher[8] says that gangrene of the lung due to a fistula between the trachea and esophagus is not an uncommon termination of cases of cancer of the esophagus. He reports the history of a patient who had difficulty in swallowing for six months, but who suddenly began to cough badly and to have great increase in the difficulty in swallowing even water. He promptly died at the end of a week, and upon autopsy a large fistulous opening was found between the esophagus and trachea. This case was a unique one in Mosher's experience, and must be considered as rather unusual, in spite of the frequency with which cancer involves the esophagus and upper extremity of the trachea and larynx.

The interest attached to a study of these cases, whatever the underlying cause, is in the aid rendered by modern methods in confirming the diagnosis and in the hope that by one of these methods the lesion, when due to other than an incurable affection, may be remedied by some surgical procedure. Unfortunately, the majority of cases of fistula between the esophagus and trachea have been due to carcinoma or tuberculosis. A few cases, however, of less fatal diseases, including syphilis, and some in which the underlying cause could not be determined, have been recorded. In all of the reported cases the similarity in the symptomatology and prognosis to that found in the congenital variety was such as was to be expected. Nevertheless, it is of interest to review the essential features of some of them.

Heddaeus[9] reports the case of a male, aged fifty-eight years, in whom there were found tuberculosis and enlarged bronchial glands. These had undergone suppuration, resulting in a necrotic opening in the anterior wall of the esophagus and posterior wall of the trachea, about the size of the end of one's little finger. The pathologic findings were sufficiently doubtful to raise the question of carcinoma, in addition to the above.

The cases of Riviere[10] were those of children in whom enlarged and caseous glands were associated with tuberculosis of the lungs and intestines. The glands had become adherent to both esophagus and trachea, and the perforation occurring from the trachea to the esophagus permitted the gradual ingestion of tuberculous secretion by the stomach and intestines, thus developing, in addition to the glandular tuberculosis, tuberculosis of the intestines.

Pachina,[11] on the other hand, believes that fistulas are due mostly to tuberculosis of the esophagus, the penetration being from that tube to the air passages. He reports a case in which four to five centimeters above the cardia, in the left wall of the esophagus, there were two openings leading into lung cavities.

An exceedingly interesting report is that of Dorner,[12] in which an opening was found between the left bronchus and the esophagus, due to necrosis from the pressure of an aortic aneurism. The aneurism in this instance did not burst, as has been the case in others, but pressure necrosis of both esophagus and bronchus caused the fistula. He quotes Ewald, to the effect that fifty per cent of all the esophagotracheal fistulas are due to cancer of the esophagus. Among other causes, he mentions mediastinal tumors, tuberculosis, gumma and traumatism.

The case of Mouisset and Mangini[13] is of interest because of the valuable aid given by tracheoscopy. In this case there was difficult breathing, but a useless tracheotomy was avoided by finding the cause through the employment of the tracheoscope.

Basch[14] reports a case in which the patient had received an injection of salvarsan for gumma of the nasopharynx and pharynx. Twenty-four hours after the injection the patient died. The necropsy showed putrid bronchitis and gangrene of the lungs from tracheoesophageal fistula.

In a study of penetrating wounds of the neck, Henrichsen[15] showed that although the ordinary suicidal wounds are commonly made in the upper portion of the air and food passages, rare instances are found in which the trachea and esophagus are injured.

Moritz[16] relates the case of a woman, thirty-six years old, who died from a perforation between the esophagus and trachea. Tuberculosis was excluded as a cause, and although the evidence was not entirely conclusive, the author believes the lesion to be due to syphilis. Although the usual syphilitic ulcerative lesion results in the formation of cicatrices with stenosis, nevertheless syphilis may rarely produce a perforating ulcer.

Ponzio[17] reports the case of a man, sixty-three years of age, suffering from cancer. A fistula between the right bronchus and esophagus was diagnosed by the X-ray.

Schütze[18] made the diagnosis by means of the esophagoscope and X-ray. In this he was aided by the expectoration of colored frothy sputum after the patient had swallowed a solution of methylen blue. The cause of the fistula in this case was doubtful. The question was between gumma and carcinoma.

Syphilis was probably the cause in a case reported by Dufour.[19] The patient was a woman, thirty-eight years of age. Death resulted from double bronchopneumonia, and the necropsy showed an ulceration upon the posterior wall of the trachea with fistula. Tuberculosis was excluded.

Pehu[20] relates the case of a bronchoesophageal fistula due to glandular involvement following tuberculous pericarditis.

An interesting case was reported by Martens[21] before the Society of the Charite Physicians, in which a patient, who had tuberculosis with empyema, had been operated for the latter. Later there developed an esophageal fistula opening in the back and ten bronchial fistulas. The patient remained in excellent general condition. The esophageal fistula required a tampon bandage every second day. Fortunately, there was no direct communication between the esophageal and the bronchial fistulas.

Woods[22] relates the case of a woman who had swallowed an artificial denture which had been allowed to remain in the esophagus for four months because of advanced pregnancy. A fistula was found from the gullet to the trachea. The denture was removed by esophagotomy. In the report of this case nothing was said of the subsequent history, although Woods says that the question now was how to treat the case, as the fistula still existed. In the discussion of this case, Dundas Grant thought that a large tracheal incision would be a good plan to reach the fistula, and called attention to a case reported in which a Hahn's sponge canula with a jacket on it of thin India rubber had been introduced and worn for a year, and in which the feeding could be done in comfort.

These cases were reported very briefly in the *Journal of Laryngology,* 1904, which accounts for this incomplete reference.

In 1905 de Navratil[23] gave the results of experiments upon dogs, in which he attempted an operative procedure for the closure of esophagotracheal fistula. He presented two operations, one of which gave very encouraging results and reminded one of the reference made by Dundas Grant in the discussion of Woods' case as given above.

A recent case reported by Beeler[24] shows the value of X-ray in the diagnosis. His patient died of pneumonia. The cause of the fistula was ascribed to syphilis. Unfortunately, no necropsy was obtained.

The case reported by White[25] is also instructive as showing what can be done by modern surgery. While White's case dealt with a fistula in the larynx, his method of procedure might be modified to meet the indications when the artificial opening is found lower down.

The case that I wish to report may be briefly stated as follows:

Mrs. P., aged twenty-four years, had suffered from severe asthma and bronchitis for a period of one year. There was a history of asthma in the grandfather, but no family history of tuberculosis. The patient gave no history of syphilis, being the mother of a healthy six-month-old child. She had had one miscarriage during an attack of measles. For seven months prior to her visit there was almost continual wheezing, shortness of breath, cough and expectoration. During the past year she had lost thirty-five pounds in weight. Six days before my examination the patient found it difficult to swallow both solid and liquid food. Every attempt caused violent choking and coughing. The difficulty in swallowing was sudden in its onset.

Upon examination an extremely emaciated woman with fever and rapid pulse gave one the impression of a patient suffering from tuberculosis; in fact, it was because of the suspicion that she was suffering from laryngeal tuberculosis that she was referred to me by Dr. Hoel of Fort Collins. Examination of the pharynx and larynx was entirely negative. Upon asking the patient to swallow a little water, a violent coughing attack supervened. Careful observation easily revealed the fact that the cough was produced after the water had passed the pharynx, a sufficient length of time elapsing from the moment the water was taken in the mouth to the time of the attack to give one the definite conviction that it had passed into the air passages after its entrance into the esophagus.

The physical examination of the patient by Dr. J. N. Hall showed moderate dullness at the right apex, with lessened voice and lessened respiratory murmur at times only. Abundant rales were found in the left lung, and there was rough breathing everywhere. The abdomen was negative. There was no edema, no evidence of aneurism or syphilis, and no enlarged glands. The impression given Dr. Hall was also that of tuberculosis, although repeated examinations failed to reveal the presence of tubercle bacilli.

The diagnosis of perforation between the esophagus and trachea or bronchi was confirmed by the X-ray. A bismuth meal was given, and the radiogram taken immediately. The bismuth was seen passing from the esophagus to the trachea. (Figure 1.) After a few violent coughs another radiogram was taken, and the bismuth was seen to be scattered through numerous bronchi of the left lung. (Figure 2.) The extremely weak and hopeless condition of the patient, as well as the certainty of the diagnosis, obviated the necessity of a bronchoscopic examination. Death followed in twelve days.

The necropsy was performed by Dr. Hillkowitz, who reported as follows:

The thoracic viscera were removed en masse. There were no adhesions on either side to the pleural cavity. Pericardium, heart and valves showed nothing noteworthy.

Lungs.—The lower lobe of the right lung, its posterior aspect, showed three small abscesses the size of a pea. The rest of the visceral surface on both sides was perfectly smooth. On section both lungs showed numerous minute grayish spots sprinkled through their substance.

Esophagus.—This was split open throughout its length through its posterior wall. The mucous membrane was smooth and showed no congestion. At one point, the exact location of which is given later, there was a small perforation into the trachea. This aperture was round, with smooth rounded margins, and about one centimeter in diameter. On slitting the trachea through its anterior wall, the perforation was seen about two centimeters above the bifurcation, and in line with the left bronchus. The mucous membrane of the trachea was red, congested, rough and presented numerous elevations and depressions. In two or three places the depressions were about five millimeters in diameter, and presented a distinctly puckered appearance. These were doubtless scars. The mucous membrane in the neighborhood of the perforation was highly congested, the tissue disintegrating readily on touch.

Nothing noteworthy was seen in any other organ.

Microscopic Examination.—Sections from the lung showed the alveoli filled with desquamated epithelium, red blood corpuscles and leucocytes. There were small round cell infiltrations in the interstitial tissue, as well as around the bronchi.

Sections from the mucous membrane of the trachea at the site of the scars revealed a partial denudation of the epithelium and an extensive round cell infiltration of submucous and muscular tissue. The round cells were distributed everywhere, forcing their way between the muscular tissue fibers. The blood vessels were highly congested.

Neither in the sections from the trachea nor of the lung could tubercles or giant cells be demonstrated.

Pathologic Diagnosis.—Bronchopneumonia. Perforation between trachea and esophagus by severe ulcerative process in the trachea, the etiology of which is obscure.

The cause of the perforation in this case must remain in doubt. The history of the patient and the gross physical findings before death favor tuberculosis. This cannot be confirmed because of the negative laboratory and postmortem findings. The diagnosis of syphilis is negatived by the history of the case. Unfortunately, a Wassermann reaction was not made. Postmortem findings, as revealed by the healed ulcers in the trachea, the scar tissue and the final ulcerative perforation, speak strongly in favor of syphilis as the underlying cause.

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FIGURE 1.

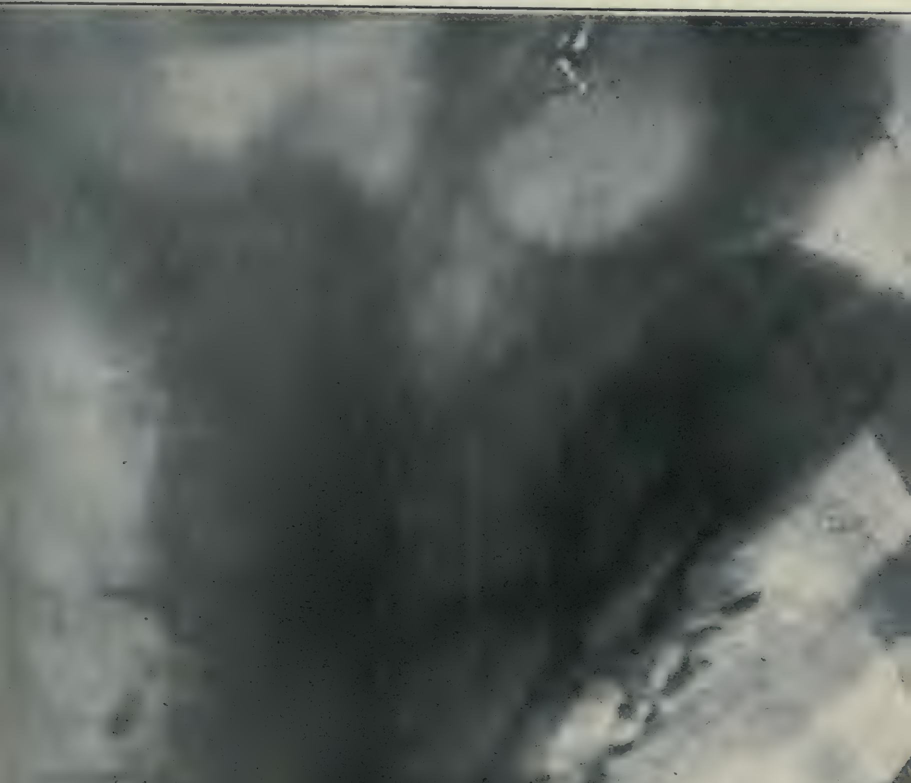

FIGURE 2.

XXXVIII.

AN EPIDEMIC OF A SEVERE FORM OF ACUTE INFECTION OF THE THROAT, WITH ABSCESS FORMATION—REPORT OF FIFTY-EIGHT OPERATIONS.

By Clement F. Theisen, M. D.,

Albany.

During the past winter, particularly in December, January and part of February, the writer was fortunate in being able to observe an epidemic of acute infection of the throat occurring in Albany, and to a smaller extent in some of the neighboring cities. Altogether three hundred and eighty-four cases came under the writer's personal observation. Nearly all the cases were treated in Albany. A few were seen in consultation in Schenectady, Troy and Rensselaer. Some of the physicians in those cities stated that they had treated a large number of cases, but the writer was not able to secure any accurate data as to the exact number.

Of the total number of cases observed, three hundred and eighty-four, fifty-eight required surgical measures. Forty-four were cases of peritonsillar abscess, and in nearly all these cases diseased tonsils were present; in the majority of the submerged variety, with crypts high up in the upper lobe of the tonsil. Thirty-eight cases were unilateral, and in six, abscesses developed on both sides. Of the remaining fourteen cases requiring operation, in eight the abscess developed in the lateral columns of the pharynx, in two well up in the nasopharynx. There were two cases of infection of the epiglottis with great edema and some pus, two cases of abscess of the lingual tonsil, and two of retropharyngeal abscess, the last two occurring in children. It is worthy of note that in this series of fourteen cases, in ten there were no enlarged tonsils, and in the other four, healthy looking tonsils with no deep crypts. In the cases then in which the infection did not originate in the tonsils, and in which no diseased tonsil tissue could be demonstrated, the infectious process selected other lymphoid structures like the lateral columns and the lingual tonsil.

We could not determine that the cases in this epidemic answered the typical description of epidemics of socalled "septic sore throat," nor could the streptococcus of Smith, which is supposed to be the organism responsible for septic sore throat, be isolated.

I suppose all cases of acute infection of the throat, with abscess formation, might properly be called septic, irrespective of the particular variety of streptococcus isolated in the cultures.

The writer has come to the conclusion that Semon's opinion, stated in his paper on the probable pathologic identity of the various forms of acute septic inflammation of the throat and neck, and since then in other papers, is the correct one.

Semon believes "that the various forms of acute septic inflammation of the throat and neck, hitherto considered as so many essentially different diseases, are in reality pathologically identical; that they merely represent degrees varying in virulence of one and the same process." It is known, for example, that a certain variety of the pneumococcus will produce localized edema. Welch, in his paper on the micrococcus lanceolatus, has determined by animal experimentation that this particular kind of pneumococcus or diplococcus, when injected subcutaneously, produced extensive local edema. This is a common observation also in cases of peritonsillar abscess and other forms of streptococcus angina, so that from a clinical and practical point of view it does not matter much what particular variety of the streptococcus is responsible for epidemic infections of the throat. The pathologic changes in the tissues are much the same.

Personally the writer is frank to admit that he has not been able to find the special distinctions and differences in the appearance of the throat, in epidemic sore throat, such as have been described by Smiley, who, working with Smith, investigated epidemics occurring in Boston, in which they isolated the particular variety of streptococcus now known as the streptococcus of Smith.

The epidemic in Albany was at its height during December and January; after the middle of February very few cases developed. During December and January the writer was called to see new cases daily. These occurred in all parts of Albany. We did not think that the epidemic was caused by infected milk, as the families afflicted got milk from many different dairies and milkmen. The cases of sore throat were distributed, as above stated, in all parts of the city, no greater number of cases occurring in families supplied by a particular dairy. During the worst of the epidemic so many new cases developed every day that we did not have time to study them carefully. The cultures that were taken in the beginning practically showed the streptococcus as the predominating organism. After that, as the cases in which no abscesses developed were so similar clinically, no cultures were taken, except in cases in which a distinct membrane was present, or when the diagnosis was in doubt. Patients were all very ill from the very onset of the infection, many of the cases starting with chills. In nearly all there was great prostration, and in the cases occurring in children, very high temperatures were the rule. Joint complications, acute arthritis and polyarthritis occurred in twelve of the cases, acute endocarditis in one case, and in twenty-four, examinations of the urine showed the presence of albumin and casts.

In the cases occurring among children between the ages of six and fourteen, eighteen in number, acute otitis media, requiring incision of the tympanic membrane, developed in eight. These cases were all clinically follicular tonsillitis, and could not be distinguished from the usual sporadic cases.

There were sixty other acute ear complications occurring among the cases during the epidemic. In the majority of cases the writer was called in time to incise the tympanic membrane very early, and this was undoubtedly the reason that no mastoid complications developed. There was only one mastoid, and that in a child, aged fourteen years. In this case the drum membrane had ruptured ten days before the writer was sent for.

It would make this paper much too long to report cases in detail, so all the cases of peritonsillar abscess will be grouped and considered as a whole.

The forty-four cases of this kind occurred in patients between the ages of ten and seventy-two years, the majority occurring in young adults betwen twenty and thirty years of age. There were four cases in children, three girls and one boy, aged twelve, ten, thirteen and fourteen years. In nearly all these cases, as before stated, the tonsils were found to be enlarged and abnormal in other respects. Thirty-eight cases were unilateral and six bilateral. A number of the patients between fifty and seventy years of age had not had any trouble with their throats for years, and came down with quinsy in this epidemic.

The abscess cases started very acutely, in many the usual symptoms being much intensified. Abscesses developed rapidly, with great edema of the surrounding parts, and swelling of the glands of the neck, particularly in the bilateral cases. In two of the bilateral cases there was great difficulty in breathing, due to an extension of the edema to the laryngeal mucosa. The constant use of an iced adrenalin spray afforded the greatest relief, and was continued after the abscesses were opened. Arthritis occurred in eight of the abscess cases, and endocarditis followed one case. There were four other cases of arthritis occurring in cases in which abscesses did not develop. Examinations of the urine were made only in the abscess cases, and in twenty-four albumin and casts were found.

In the management of the cases of peritonsillar abscess, I would like to mention a method that was employed with great success.

We have all experienced the difficulty in operating on these cases after they have been going on for a number of days, the patient at times not being able to separate the teeth far enough to admit even the handle of a teaspoon. If the cases are operated on early, this difficulty is done away with. Pus is present in the majority of the cases and can be reached after forty-eight hours.

For several years I have been following a method suggested by Ballenger—that is, a dissection backward toward the capsule until the pus is reached.

In fourteen of the cases operated on during the epidemic last winter, I have gone a step farther and have removed the upper lobe of the tonsil which had been loosened by the dissection. This leaves a large triangular opening for drainage. I have not found, and I agree with Barnes in this, that operating in this way during the acute stage, there was an increased chance for a more extensive infection. The entire sinus tonsillaris is usually infected in these cases, and patients so operated upon have done much better, and recovered more quickly than the ones treated by the usual incisions.

Barnes in his paper on the radical treatment of peritonsillar abscess by tonsillectomy, speaks of the difficulties of locating the pus when it is confined between the body of the tonsil and the superior constrictor muscle, and recommends dissecting out the tonsil in this class of cases. He has reported excellent results in ten cases.

It is unfortunate that we are frequently called to see cases of peritonsillar abscess so late, and when the edema on the affected side is so great that it is difficult and in some cases impossible to find the usual anatomic landmarks. In such cases the operative procedure described above was not attempted. It was performed only in selected cases.

The abscesses developing in the lateral pharyngeal columns are of particular interest, and are easily overlooked. The writer's attention was called to this class of cases particularly by Swain's paper on the subject. In a very instructive paper Swain reports several cases. In one, where he was able to make a thorough examination of the throat after the rupture of an unsuspected abscess of the lingual tonsil, a subsiding swelling of the epiglottis and aryepiglottic fold could be seen. The abscesses in the cases of this type occurring during the epidemic in Albany developed slowly, and for a number of days the writer could not determine the cause for the great distress and pain in swallowing that the patients complained of. In six of the cases the abscess was discovered high up in the lateral pharyngeal wall, the marked localized swelling finally calling my attention to the source of the trouble. In two other cases the soft palate was pushed forward. Pus was evacuated in all after the first deep incision. In the two retropharyngeal cases the bulging was found almost in the median line of the posterior pharyngeal wall.

Local anesthesia was employed in all cases, except in a few of the peritonsillar abscess cases in children, as the writer is of the opinion that general anesthesia is not entirely safe in this class of cases.

Roe, in his very complete paper, "Phlegmons of the Upper Respiratory Tract," reported two fatal cases of abscess of the throat. In the first case, that of a young man, aged twenty years, a retropharyngeal abscess developed in the right side of the throat, which was opened. In spite of all efforts to save his life he died of exhaustion in three days. The throat infection was mixed—streptococci predominating. In the second case, a young woman, aged eighteen years, the throat was very much swollen, finally involving the laryngeal mucosa, and requiring tracheotomy. Patient died five days after the onset of the attack. This was also a mixed infection—pneumococcus and streptococcus.

Roe also mentions cases of faucial phlegmon in children resulting in retropharyngeal abscess. The abscesses were opened in all these cases, patients making good recoveries.

The larynx is frequently involved in acute pharyngeal infections, particularly when abscess formation occurs. The great edema of the surrounding tissues that frequently accompanies peritonsillar abscess and abscesses in other parts of the pharynx may extend to the laryngeal mucous membrane. Some of these cases are fatal.

Goodal has reported thirty-five cases of a very virulent form of ulcerative sore throat, twenty-five of which were fatal. The principal lesions were in the pharynx, but in nine the larynx was also involved. In these severe septic and ulcerative throat conditions the streptococcus is the organism most frequently demonstrated. Undoubtedly more virulent forms of streptococci are responsible for the serious complications, such as arthritis, endocarditis and nephritis, occurring with such throat conditions.

In the epidemic form which has been reported from many parts of this country and England the throat infection appears to be milder than in the sporadic cases.

In an epidemic in Baltimore, Hirschberg found, in addition to streptococci, pneumococci in forty-eight cases.

Davis and Rosenow, in the Chicago epidemic, found a peculiar streptococcus causing many serious complications and deaths. Cases had a sudden onset with severe prostration, enlargement of the glands and other complications. The authors isolated an encapsulated hemolytic streptococcus of high viruleney which sometimes coagulates milk.

Cases of mastitis were discovered in the suspected herds, and some cows that had not been suspected were discharging pus with the milk. This special streptococcus is thought to be related to the streptococcus pyogenes group which Leutscher has classified. He divided the group as follows: The ordinary streptococcus pyogenes group, which does not clot the milk or ferment raffinose. The streptococcus angiosus group, which often clots milk and ferments raffinose. He states that the streptococcus pyogenes group is usually associated with suppurations, erysipelas and septicemia, and that the streptococcus angiosus group is particularly associated with inflamed throats, and with malignant endocarditis and otitis.

A mild epidemic of sore throat occurred in Schenectady in 1912. Van der Bogart observed sixty-three cases, of which thirteen occurred in infants and thirteen between the ages of ten and fifteen years. It could be positively determined in this epidemic that the milk supply was not responsible in any way.

The ordinary run of the cases in the Albany epidemic, excepting the cases where abscesses formed, was characterized clinically by the intensely acute angina, great pain in swallowing, glandular swelling in many cases, extreme prostration and weakness, and unusually long continued temperature. The onset was usually very sharp, often starting with a chill and sharp temperature elevation. The writer was fortunate in having no deaths among his cases, although a number of the patients were seriously ill, and two just escaped tracheotomies.

In the treatment of the abscess cases after operation, patients received great relief from the use of frequent irrigations of the throat with normal salt solution and Seiler's solution. A fountain syringe was used, care being taken to control the flow so that a gentle current of fluid, under practically no pressure, was carried into the throat.

A study of the reported epidemics of septic and other forms of epidemic sore throat proves fairly conclusively that in the majority the milk supply was largely responsible. It is now generally known and believed that they are caused by a hemolysing type of streptococcus transmitted by the milk.

We can readily understand that milk as well as other body fluids may favor the development of a pathogenic power in the streptococcus.

In an epidemic at Rockville Centre, Long Island, milk was collected separately from each quarter of the udder of each cow. One cow showed mastitis, and streptococci isolated were bovine in origin. In one quarter with many streptococci the milk was flocculent. Both hemolyzing and nonhemolyzing strains were obtained, but few had the fermentative character of the bovine strain. The conclusion was reached in this epidemic that the cows were infected by the milkers.

Mellen, in his paper on the early diagnosis of streptococcic sore throat and the effects of vaccine in its treatment, comes to the following conclusions:

Early examination is valuable, as it gives a clue to the virulence of organisms.

It is not necessary to have symptoms for an individual to carry the organisms.

The acute cases respond quickly and decisively to a vaccine of proved immunizing power, if it is used early.

The condition should not go untreated, even though in most cases it is self limited, as it has many sequelæ.

Even after the symptoms are allayed, the body is still apt to absorb a few remaining organisms, with sometimes serious results.

Rosenow and Moon reported an epidemic of sore throat in Elmhurst, Illinois, occurring in February, 1914, which could be traced to the milk—isolated virulent streptococci from the milk.

Thirty cases used milk from the same dairy, which drew milk from eight farms.

Seven ounce samples of milk were centrifuged at high speed for twenty minutes. Sediment obtained was streaked in blood agar and inoculated into ascitis dextrose broth. Similar streptococci to the ones found in the patients' throats were isolated in nine out of eleven samples. They produced hemolysis on blood agar, and of them four were virulent for guinea pigs. On the use of pasteurized milk the epidemic subsided.

CONCLUSIONS.

1. The epidemic subsided when pasteurized milk was used.
2. Virulent streptococci showed preference for certain structures.
3. Organisms from milk resembled rheumatic strains culturally.
4. Involvement of muscles and joints occurred in patients infected by milk.

North, White and Avery, in a study of an epidemic of septic sore throat in Cortland and Homer, New York, in April, 1913, traced the source to milk. In three days a widespread epidemic developed. Mastitis was found among the herd in the dairy supplying most of the milk to both towns. The following results were obtained:

1. Seventy per cent of the cases in each town occurred among people taking milk from the same dairy.
2. Mastitis was found in two cows of this dairy herd.
3. Streptococci found in patients' throats were identical to those found in the milk from cows with mastitis.
4. The streptococci were similar to the ones found in cases of mastitis in cows of another herd.

At Finckley, England, in 1904, streptococci were demonstrated in throat cultures. In this epidemic five hundred and fifty cases were reported.

At Guildford, England, in 1903, streptococci were detected in the suspected milk.

At Christiania, in 1905, this was also found.

Mastitis in cows was noted in all four of these outbreaks, and the organisms were a hemolytic capsulated type. The cases appeared in winter and spring.

Symptoms.—Sudden onset, with sore throat, intense hyperemia and grayish exudate. Enlarged cervical glands with some suppuration was common. Extreme prostration was characteristic. The complications were otitis media, peritonsillar abscess, erysipelas, nephritis, endocarditis and myocarditis.

The infection was traced directly to the milk supply.

Streptococci were demonstrated in the mixed milk of the entire herd at a suspected farm.

Mastitis was found in the cows of this herd.

Streptococcic sore throat was diagnosed in the milkers.

Streptococcic sore throat was seen in other employees who handled the milk.

King has recently reported a series of cases of severe infection of the tonsils, followed by arthritis, endocarditis and other complications, in which the Conellan-King diplococcus was found in the crypts of the tonsils.

Seventy-eight cases have been observed by the author, in all of which this diplococcus was found.

In conclusion, I would like to give brief histories of two cases, one of abscess of the socalled lateral column of the pharynx, and the other an abscess of the lingual tonsil. Both occurred in young patients, aged eighteen and twenty-one years.

Case 1.—L. F., a young man, twenty-one years old. Was called to see him in December, 1915. He stated that for a couple of days he had had an extremely severe sore throat, starting with a chill and sharp temperature elevation. His physician, who called me to see the case, said he had been subject to sore throats. On inspection the entire pharyngeal mucous membrane proved to be intensely congested and reddened, the tonsils, however, not being particularly involved. Epiglottis and larynx appeared normal. I stated that it was an ordinary acute angina, and prescribed ice and an astringent gargle. His difficulty in swallowing got steadily worse, and I was again asked to see him at the end of another forty-eight hours. The throat looked much the same, except that high up on the right side of the lateral wall of the pharynx, above the arch of the soft palate, there was a decided bulging which pushed the soft palate forward. A deep free incision was followed by the escape of a rather thick pus. His recovery from this time was uneventful.

The second case, that of a young woman, eighteen years of age, started in a similar manner with a chill and temperature elevation. I was not asked to see this case until the throat symptoms had existed for five or six days. She was having some difficulty in breathing and great difficulty in swallowing. The right side of the fauces was very edematous, and examination with a laryngeal mirror disclosed the seat of the trouble at once. The lingual tonsil was enormously swollen and very edematous, so much so indeed that the larynx could not be seen. This was of course responsible for her difficult breathing. The first attempt, a deep puncture failed to locate the pus. We then cocainized thoroughly and made a deep incision. This was followed by a very profuse hemorrhage and some pus. The bleeding was finally controlled by ice in the mouth and an ice bag externally.

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XXXIX.

ACUTE MASTOIDITIS WITH UNUSUAL SYMPTOMS INDICATIVE OF INTRACRANIAL INVOLVEMENT—OPERATION—RECOVERY.

By Edward Bradford Dench, M. D.,

New York.

The patient was a young woman, aged seventeen years, who, following an attack of grippe, suffered from an acute otitis. She was seen by me on the eighth day after the inception of the middle ear inflammation. During this period two myringotomies had been performed. The night before I saw the patient the temperature had risen to over 103°, and on the day that I saw her the temperature was 104°. She presented the usual signs of a narrowed fundus of the external auditory meatus and a bulging drum membrane. The mastoid was exquisitely tender throughout. An immediate operation was performed. The mastoid cells were found to be filled with blood and some free pus. The mastoiditis was clearly of the hemorrhagic variety. A complete operation was done, and the dura in the middle fossa was exposed over an area about three-quarters of an inch square. The dura appeared healthy. The zygomatic cells were exceedingly well developed, and these were thoroughly eradicated. The upper portion of the wound was sutured and drained from its lower angle by means of two gauze drains. Owing to the high temperature, an immediate blood culture was made, and this was repeated on the second day. Both blood cultures were negative. The patient's temperature, at the time of operation, was 104°, and it fell with regular remission until about the fourth day after operation it became normal and so remained.

The patient left the hospital about ten days after the operation. She seemed very weak, and although she complained of no pain at this time she did not seem to be doing well. Two weeks and a half after the operation the patient began to have headaches. The pain was located chiefly in the frontal and temporal regions. This pain was so severe as to keep her awake. Three weeks after the operation the patient came to the office with a temperature of 104° and a pulse of 142. She was immediately sent to the hospital and a blood culture taken. This was negative. The temperature gradually fell to normal, and the wound, which throughout had been doing poorly, seemed to improve.

The patient left the hospital again in about a week, and at this time had very little pain. One month and three days after the primary operation the headache returned and there appeared a paralysis of the external rectus oculi of the corresponding side. The patient at this time was suffering from severe frontal headache, was excessively tender to pressure over the first branch of the trigeminal nerve, was pale and anemic, and was exceedingly hysterical and lacrimose. Careful tests showed no ataxia, no nystagmus, fair hearing in the operated ear, a normal temperature and a normal pulse. Although the site of operation was the left side, there was no aphasia. The patient was given bromids, internally, to relieve the extreme nervousness from which she was suffering. These symptoms continued, with increasing severity, for about twelve days. During this time the abductor paralysis became absolute. The temperature and pulse during this period remained normal. The patient's general condition, however, steadily deteriorated. She became more and more nervous, the pain in the head increased, she lost flesh, and her progress was in every way unsatisfactory.

Six weeks after the first operation the patient again entered the hospital—on account of the severe headache and her general nervous symptoms. At this time the abducens paralysis was complete, but aside from this there was absolutely no evidence of any intracranial involvement. The hearing was fair on the affected side, there was no spontaneous nystagmus, and no evidence of any labyrinthine involvement. There was no aphasia, and an ophthalmoscopic examination showed both optic discs perfectly normal. Spinal fluid negative. The patient was seen in consultation with Dr. Pearce Bailey, who agreed with me that there were no signs of any intracerebral involvement. While the case resembled those cases first reported by Gradenigo, I felt certain that there was no deeper focus of suppuration, owing to the thoroughness of the original operation. I could only explain the pain by assuming that there had been an inflammatory process extending along the dura from the area of original exposure inward to the Gasserian ganglion, and that this inflammatory process accounted for the pain. Such an inflammatory process would naturally account for the abducens paralysis.

One month and two weeks after the first operation I reopened the wound and extended the incision forward to a point one inch behind the margin of the orbit. The dura of the middle cranial fossa was exposed over an area about three inches in diameter. This area included the area of dural exposure at the primary operation and extended somewhat posteriorly to this; in other words, the dura of the entire middle fossa was exposed. The dura was carefully elevated by means of a flat spatula, and the entire floor of the middle fossa explored, including, of course, the roof of the petrous pyramid. Absolutely no pus was found. The dura at the site of original exposure was somewhat thickened, but otherwise the dura appeared to be perfectly normal. Firm dural adhesions at the anterior extremity of the first area of dural exposure were broken down by means of the blunt dissector. A folded rubber tissue drain was introduced to the very apex of the petrous pyramid. The anterior portion of the incision was closed and the mastoid wound was left wide open. I wish to emphasize here that absolutely no pus was found in either the mastoid wound or the middle cranial fossa. In other words, the first operation had been absolutely thorough, and this portion of the wound required no revision. The day after the operation the patient's temperature rose to 105°. She complained of no pain, however, and was perfectly rational.

From this time on she began to improve, and ultimately made a complete recovery. On the second or third day after the operation she developed beginning choked discs in both eyes. This swelling of the optic papillæ gradually increased and was more marked upon the operated side. The rubber tissue drain was removed from the middle fossa, and immediately the optic neuritis began to disappear.

The subsequent history of the patient is one of uninterrupted recovery. The wound healed perfectly, the hearing on the affected side was excellent, and the abducens paralysis had practically disappeared when the patient was last seen. Subsequent reports of the patient show that she is in a practically normal condition.

Perhaps the most comprehensive article on this class of cases was written by Dr. Chas. E. Perkins, and published in the ANNALS OF OTOLOGY for September, 1910. The case which I have reported, however, presents some new points, and is particularly interesting from the fact that at the time of the second operation no extradural area of suppuration was found, neither was any deep focus of pus found within the bone, as so frequently occurs in these cases. In a number of cases reported the mastoid cells were found to extend into the apex of the petrous bone, and an evacuation of pus in this region was followed by immediate relief. In the present case the pain seemed to be due simply to a low grade of inflammation of the dura extending inward and involving the Gasserian ganglion. The sixth nerve was also involved as it crosses the apex of the petrous pyramid. The stripping up of the dura from the underlying bone seemed to cause a resolution of the inflammatory process with a subsidence of all the symptoms. The case is particularly interesting as bearing upon trigeminal neuralgia in cases of middle ear suppuration. The case certainly demonstrates that severe intracranial symptoms may occur without any demonstrable focus of pus in cases of this character.

XL.

THE ORAL CAVITY IN RELATION TO EAR, NOSE AND THROAT.*

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The oral cavity, located as it is at the beginning of the alimentary tract, and in the midst, it may be said, of the respiratory system, cannot but be most important pathologically, as it has been recognized to be physiologically.

Until recently the medical profession has ignored this very important cavity, leaving it entirely to the dentist, whom, while not considered competent, as he was said not to be a pathologist, was not only left in possession, but what he said or advised in individual cases was ignored or scoffed at.

The writer cannot, at this time, take up the matter of education of either the dentist or the physician, but suffice to say that he has long been of the opinion that the dentist should know more about the body in general, and the physician should know a great deal more about the oral cavity. However, under present conditions, they may each gain much by observation, not in the spirit of criticism of the other.

Upson expresses the feeling of the medical profession of a decade ago, as follows: "There seems to exist among physicians not only a disregard, but a distinct, though mild, dislike of the teeth as organs to be reckoned with medically; they being, as it were, an Ishmael not to be admitted to their pathologic birthright."

Lauder Brunton's essay on the subject is too little known and heeded, and few such systematic attempts have been made to correlate their disorders with the sufferings of the human race, except for the most obvious phenomena of pain. Ordinary pain, at a distance, such as headache or neuralgia, due to the teeth, though well known, is commonly disregarded. Even the various reflex phenomena in children—convulsions, fretfulness and fever—are not now ascribed to irritation, either of teething or dental caries, but to digestive disorders. The state of recent opinion, as enshrined in epigram, is that the result of teething is nothing but teeth.

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Although there has been some change in opinion in the last decade, there is yet too much tendency to laugh at what were the fears of earlier writers in medicine, and while we are able to disprove a few of their assertions, we must remember that we are as likely to make mistakes as they. In not keeping this in mind, we frequently go to the opposite extreme and miss the grain of truth, for such there is in the great majority of such writings. Furthermore, we hail with delight many new observations, new operations, etc., when they are simply stolen entire from other works.

The otolaryngologist has been interested for some time in the work of the orthodontist, on account of the change produced in the nasal cavity by the mechanical rearrangement of the teeth. It was the author's fortune to read probably the first paper on this subject presented to the medical profession, but unfortunately it was never published, but you are probably familiar with many of the other papers published on this subject.

There is nothing especially new in the line of reflex irritation of the nose, throat and ear, caused by pathologic conditions of the oral cavity, unless it is that produced by the condition known as pyorrhea, as this pathologic entity has been more recently considered by the medical profession from the standpoint of its effect upon, not only the gastrointestinal tract, but often the urogenital tract and ductless glands and joints, as well as the circulatory system.

In order to fully understand the reflexes, we must review our anatomy of the head, and especially that of the fifth nerve, with its connections through its ganglia with motor and

The relation of the oral cavity to the system in general has been given a great deal of attention by the physician in the last two years. Whether the infection always takes place through the gastrointestinal tract, as from the swallowing of pus, or whether by absorption through the lymphatics surrounding the jaws, we must probably take the same view that we do in regard to the tonsils—that while a certain amount of the toxins may be introduced into the system in this way, the greater portion is discharged into the lymphatics or blood systems.

In a case of pyorrhea alveolaris we have a nearer approach to the tonsil than in any other condition in which the teeth are concerned, while abscesses in individual teeth resemble an abscess in any other portion of the body.

Whether the secretory function of the glands discharging into the oral cavity is affected by infection in that cavity more than by infection in other portions of the body, is a question that would probably be answered in the negative, with the exception of cases where inflammation, confined to these secretory organs themselves, is found.

Beck's formula of percentages of infection gives us something to work from, and we believe is correct in the early years of life:

From tonsils	20 per cent
From gastrointestinal tract	10 per cent
From appendix	10 per cent
From gall bladder and several other recesses	10 per cent
Total degree of infection	50 per cent
Resistance	50 per cent

These should be revised after the twentieth year, decreasing the percentage of infection from the tonsil by at least ten per cent, and increasing that of the gastrointestinal tract by the amount of decrease from the tonsil, the percentage of the increase in the gastrointestinal tract being credited to the teeth. It must be kept in mind that all infection from the mouth does not necessarily travel through the three routes above mentioned, but that in many cases, especially in the chronic mouth breather, infection of the larynx, trachea, and bronchi may take place in cases of aspiration of the infected material. However, these structures are probably saved from infection somewhat because of the fact that the mouth very soon becomes dry and the dried mucus on the tongue, lips and pharynx prevent the bacteria from being aspirated, as they would be if the parts were kept continually moistened.

While the general surgeon and the internist are looking for the origin of diseases treated by them in remote parts of the body, and while many of these have been traced to the oral cavity, does it not behoove those of us who are working in the regions surrounding this cavity to examine it more closely for the origin of some obscure condition which we are called upon to treat? How easy it would be for the laryngologist (and many I am glad to say are availing themselves of the opportunity) to examine the teeth and jaws before looking deeper into the throat. How many of us would attempt to treat an ear condition without first making a thorough examination of the nose and nasopharynx? And this we have known must be done for some time, while at the same time looking through the oral cavity into the throat and only seeing the tonsil diseased, many of us, I am sorry to say, not knowing whether the teeth were present or not, with the exception, possibly, of the six anteriors. We could derive a great deal of benefit by having a dentist associated with us on the staff of each hospital; not the old time dentist, but the modern dentist, who knows not only how to fill and extract teeth, but who knows the pathology of conditions found in the oral cavity; who knows how to take skiagraphs and interpret same. Such a man should be of inestimable value to the laryngologist, and also to the internist and surgeon.

Few articles have been written on the subject of reflexes associating the mouth with the eye, ear, nose, or throat, although this subject is mentioned in many text books—attention being first called to it by Richter in 1795, and more practically by Hilton in his book on "Rest and Pain" in 1863, as well as in later additions. All books on oral surgery, first being that of Garretson, and two valuable articles, published since 1903; the first by Kate Wiley Baldwin in the *Journal of the American Medical Association,* January 31, 1903, and one by Clarence E. Ide, in the *Laryngoscope,* February, 1914; although many references have been made in other articles.

We are all familiar with the toothache, or pain in the jaw, which is frequently produced by impacted cerumen in the external auditory canal, and also with the cough produced when an instrument causes pressure on the lower portion of the external auditory canal. The pain produced in the ear by ulcers on the tongue, abscessed condition in the region of the third molar or tonsil; the latter not always produced reflexly, but sometimes by direct pressure upon the eustachian tube. Ide treats this subject very thoroughly, by taking the nerves up under different heads, and we will follow him with a few slight changes.

A—Nerves of the external auditory canal and parotid gland.

1. Cervical plexus, branches of which are distributed to the hypoglossal, vagus, auricle, mastoid and parotid gland, besides branches to the sympathetic. The most important branch, auricularis magnus, arises from the second and third cervical nerves and winds around the posterior border of the sternomastoid, and after perforating the deep fascia ascends upon the muscle beneath the platysma to the parotid gland, where it divides into numerous branches.

The facial branches pass across the parotid and are distributed to the integument of the face; others penetrate the substance of the gland and communicate with the facial nerve.

The posterior or auricular branches ascend to supply the integument of the back part of the pinna, communicating with the auricular branches of the facial and pneumogastric nerves.

The mastoid branch joins the posterior auricular branch of the facial, and crossing the mastoid process is distributed to the integument behind the ear.

The occipitalis minor arises from the second cervical nerve; it curves round the posterior border of the sternomastoid above the preceding, and is directed almost vertically along the posterior border of that muscle to the back part of the side of the head. Near the cranium it perforates the deep fascia, and is continued upward along the side of the head behind the ear, supplying the integument and occipitofrontalis muscle, and communicating with the occipitalis major, auricularis magnus, and posterior auricular branch of the facial.

This nerve gives off an auricular branch which supplies the attolens auris and the integument of the upper and back part of the auricularis magnus. This branch is occasionally derived from the great occipital nerve. The occipitalis minor varies in size; it is occasionally double.

2. The auriculotemporal nerve arises by two roots, between which the middle meningeal artery passes. It runs backward beneath the external pterygoid muscle to the inner side of the neck of the condyle of the lower jaw. It turns upward with the temporal artery, between the external ear and the condyle of the jaw, under cover of the parotid gland, and, escaping from beneath this structure, ascends over the zygoma and divides into two temporal branches. The posterior temporal, the smaller of the two, is distributed to the upper part of the pinna and the neighboring tissues. The anterior temporal accompanies the temporal artery to the vertex of the skull and supplies the integument of the temporal region, communicating with the facial nerve and orbital branch of the superior maxillary.

The auriculotemporal nerve has branches of communication with the facial and otic ganglion. Those joining the facial nerve, usually two in number, pass forward from behind the neck of the condyle of the jaw to join this nerve at the posterior border of the masseter muscle. They form one of the principal branches of the communication between the facial and the fifth nerve. The filaments of communication with the otic ganglion are derived from the commencement of the auriculotemporal nerve.

It gives off auricular branches, two in number, inferior and superior. The inferior auricular arises behind the articulation of the jaw, and is distributed to the ear below the external meatus; other filaments twine round the internal maxillary artery and communicate with the sympathetic. The superior auricular arises in front of the external ear and supplies the integument covering the vagus and pinna.

Branches to the meatus auditorius, two in number, arise from the point of communication between the auriculotemporal and the facial nerves, and are distributed to the meatus.

A branch to the temporomaxillary articulation is usually derived from the auriculotemporal nerve.

Parotid branches, which supply the parotid gland, are also given off from this nerve.

The lingual or gustatory nerve supplies the papilla and mucous membrane of the tongue. It is deeply placed throughout the whole of its course. It lies at first beneath the external pteryoid muscle, together with the inferior dental nerve, being placed to the inner side of the latter nerve, and is occasionally joined to it by a branch which crosses the internal maxillary artery. The chorda tympani also joins it at an acute angle in this situation. The nerve then passes between the internal pteryoid muscle and the inner side of the ramus of the jaw and crosses obliquely to the side of the tongue over the superior constrictor muscle of the pharynx, and between the styloglossus muscle and the deep part of the submaxillary gland; the nerve lastly runs along the side of the tongue to its apex (first running across Wharton's duct), lying immediately beneath the mucous membrane. (As it passes to the side of the base of the tongue it is accessible for operation.)

Its branches of communication are with the facial through the chorda tympani, the inferior dental and hypoglossal nerves, and the submaxillary ganglion. The branches to the submaxillary ganglion are two or three in number; those connected with the hypoglossal nerve form a plexus at the anterior margin of the hpoglossus muscle.

Its branches of distribution are few in number. They supply the mucous membrane of the mouth, the gums, the sublingual gland, the filiform and fungiform papilla, and mucous membrane of the tongue, the terminal filaments anastomosing at the tip of the tongue with the hypoglossal nerve.

The inferior dental is the largest of the three branches of the inferior maxillary nerve. It passes downward with the inferior dental artery, at first beneath the external pterygoid muscle, and then between the internal lateral ligament and the ramus of the jaw to the dental foramen.

B—Nerves of the tympanic membrane.

1. The outer surface of the drum membrane and anterior and inferior portions of the canal are supplied by the auriculotemporal branch of the inferior maxillary nerve.

2.—The inner surface of the drum membrane is supplied by the tympanic branch of glossopharyngeal.

C—Nerves and tympanum: Tympanic plexus (Jacobson's), consisting of:

1. Tympanic branch of ninth cranial nerve. From the petrous ganglion passes through the temporal bone between the carotid canal and jugular foramen; ascends to the tympanum,

entering by an aperture in the floor close to inner wall. Ends in two branches which ramify in grooves on the promontory.

2. Small deep petrosal nerve. From carotid plexus of sympathetic passes through wall of carotid canal to join Jacobson's nerve. Sends branch to great superficial petrosal through inner wall of tympanum in front of foramen ovale.

3. Small superficial petrosal nerve. From otic ganglion through foramen in middle fossa of skull (or through foramen ovale), passing backward to enter petrous bone again external to hiatus Fallopii; receives a filament from the facial nerve and joins Jacobson's nerve to assist in the formation of tympanic plexus. (The inferior maxillary branch of the fifth cranial nerve sends a branch to the tympani (tensor) muscle by way of the otic ganglion. The tympanic branch of the facial nerve supplies the stapedius muscle.)

D—Nerves of the eustachian tube: Tympanic plexus (See C).

E—Nerves of articulation and mandible:

1. Auriculotemporal branch of trifacial nerve, already described.

2. Masseteric branch of inferior maxillary nerve.

F—The otic ganglion is connected with the inferior maxillary nerve by its internal pterygoid branch through two or three short filaments, and it also receives a branch from the auriculotemporal nerve. Its sympathetic root comes from a plexus surrounding the middle meningeal artery. It also communicates with the glossopharyngeal and facial nerves through the small petrosal nerve continued from the tympanic plexus. Its branches of distribution are a filament to the tensor tympani and one to the tensor palati.

The submaxillary ganglion, situated in the deep portion of the submaxillary gland, near the posterior border of the mylohyoid muscle, being connected by filaments with the lower border of the gustatory nerve. The motor root comes from the corda tympani, and its sympathetic from a plexus around the facial artery. Its branches supply the submaxillary gland, Wharton's duct and the mucous membrane of the floor of the mouth.

G—Nerves of the nasal fossa. The sphenopalatine (Meckel's) is found in the sphenomaxillary fossa, close to the sphenopalatine foramen. Its motor root is derived from the facial through the vidian. Its sensory root from the fifth and its sympathetic root from the carotid plexus through the vidian. Its branches are divided into four groups. The first or ascending group supplying the periosteum lining the orbit. It is also said to have branches to the optic nerve, cavernous sinus, and a communicating branch with both the sixth nerve and the ophthalmic ganglion. The branches of the second or descending group are distributed to the roof of the mouth, the soft palate, the tonsil and the lining membrane of the nose. The third or anterior group descends through the posterior palatine canal, passing forward, supplying the gums and communicates with the termination of the nasal palatine nerve. While in the canal it sends branches to the nose and maxillary sinus, and to both surfaces of the soft palate. Fourth, or internal branches are distributed to the septum and outer wall of the nasal fossæ.

Reflex pain from the mouth may deepnd upon an increase in number of teeth or the displacement of one or more of the normal number. The work of James and Pitts has changed our ideas to a certain extent as to the time when the permanent teeth should be erupted. As regards the temporary set, there is very little variation in number, although two teeth occasionally may be found fused, or an extra tooth may occasionally be found. With the permanent set we are much more likely to find supernumerary teeth. Bolk reported and exhibited about thirty specimens of additional permanent molars. In the Caucasian race we do not find supernumerary molars as frequently as they are found in the negro. However, we not infrequently find a supernumerary tooth in the anterior portion of the hard palate resembling a cuspid or lateral incisor. Whether any of these indicate a reversion to type or not, will not be entered into at this time. The conditions which most frequently confront us are due either to decay of the teeth, inflammation of the surrounding tissue, or abnormally placed teeth. A few cases will serve to illustrate.

Case 1.—Dr. W. B., aged twenty-seven years, consulted me in the year 1913, giving the following history: For about three years he has had severe neuralgic pains extending to the ear and over the side of the head, coming on at frequent intervals.

Ten days ago he consulted a dentist in his home city, who attempted to extract an upper right third molar, which was thought to be the cause. After one hour the operation was discontinued with the tooth loosened but still in place. On examination the tooth was found with one of the cusps broken off and somewhat loosened, and the patient informed us that his pains had been less frequent since the tooth was worked on. Under ether the bone was burred away and an exostosed tooth removed, since which time the patient has suffered no further pain, three years having elapsed.

Case 2.—Miss N., aged twenty-two years, consulted me in 1915 with her face very much swollen on the left side, giving the following history: About December 20, 1914, she noticed swelling of the face upon both sides, which she believed due to wisdom teeth, which were treated, but on account of the swelling the dentist hesitated to extract them. About January 10th they were extracted, but face continued to swell. Skiagraphs of face revealed nothing, but suppuration taking place, the abscess was opened on February 20th, and on examination of the discharge it was found to contain the ray fungus. This patient complained bitterly of pain in the ear and over the mastoid, but nothing ever developed in this region.

Case 3.—Mr. H. A., aged twenty-four years, a patient at the National Jewish Hospital. I was asked to see him on account of swelling in front of the ear and pain in the ear. He gave a history of receiving a blow upon the side of the head a few days previous. The ear was found to be perfectly normal, but a diagnosis was made of parotitis, which was subsequently changed to tuberculosis of the parotid gland. This case was reported in full in the *Laryngoscope*, October, 1914.

Case 4.—Mrs. G., aged thirty-five years, consulted me in December, 1914, complaining of pain in the right ear. The trouble was located in right lower third molar, which was extracted and pain disappeared. This tooth was badly impacted and pressing upon inferior dental nerve.

Case 5.—Mrs. S., aged fifty-two years, consulted me for what she believed to be cancer of the upper jaw and nose, as it had been under treatment for several months. On probing sinus found in the mouth, the probe encountered what apparently was the crown of a tooth, which was verified by X-ray.

Upon removal an opening was found into the floor of the nose, and the whole condition subsequently cleared up.

Case 6.—Miss H., aged seventeen years, consulted me on account of a swelling on the right side of her face, and the blocking of the right nasal passage. No pus could be found in the nose, and X-ray revealed a partially developed third molar in the right maxillary sinus, immediately above the first and second molars. A diagnosis of dentigerous cyst was made, and upon opening the sinus, a large amount of straw colored liquid was evacuated. The third molar was removed, and recovery was uneventful.

Case 7.—Dr. B., aged thirty-seven years, consulted me on February 20, 1913, complaining of a pain in the right ear. Examination of the ear showed it to be entirely normal, as was also the mouth. The following day, being unable to see me, he consulted another ear, nose and throat practitioner, who found that the patient not only complained of pain in the ear, but also of pain in the right maxillary sinus.

Case 8.—Mr. R., aged twenty-two years, dental student, consulted me in March, 1915, on account of a swelling of the neck over the larynx. On examination the neck was found very much swollen, extending up to the floor of the mouth. He gave a history of swelling at intervals, which would subside for a few days. On examination nothing was found in the larynx, and nothing in the floor of the mouth except a patulous foramen cecum. On passing a probe into the foramen cecum, it continued down the thyroglossal into the abscessed cavity. The swelling promptly subsided, and has not recurred.

XLI.

ADENOMA OF THE PALATE.

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Neoplasms of the palate are commonly of a malignant nature, usually cancerous. They arise either in the palate itself or involve it from the nose, antrum, alveolar process or pharynx. The benign growths include papillomas, angiomas, fibromas, osteomas, odontomas, lipomas, mixed tumors, adenomas and other peculiar epithelial tumors. Adenomas of the palate are quite rare, although it seems hardly possible that they are as unusual as appears from a study of the literature. Possibly the rarity of case reports is due to the fact that the tumor usually shells out readily and often no microscopic examination is made. Moreover, some observers have classified adenomas with mixed tumors of the palate, although true adenoma is distinctly different and quite easily distinguishable microscopically. The term "adenoma of the palate" has been used loosely to indicate a slowly growing benign tumor, and tumors are at times reported as adenomas with no mention of the microscopic pathology of the growth.

Paget[1] reported thirty-one collected cases of clinical adenoma of the palate. He stated that the term adenoma in these cases is of clinical value only, implying nothing as to the microscopic appearance. In most of his cases there was no account of the microscopic pathology, and when present such accounts had little in common.

Since Paget's report (1886), I have been able to collect in the literature only eight cases with a pathologic diagnosis and five cases reported as adenoma without any microscopic pathologic diagnosis. This group seems far too small to represent the actual number of cases, but the factors already mentioned may account for the discrepancy.

Two cases of rare tumors of the palate reported herein, have been seen in the Mayo clinic; the first a case of true cyst adenoma, and the second an equally rare epithelial tumor, but not a true cyst adenoma.

Case 1 (157,265).—C. K., school girl, aged sixteen years. Examined December 6, 1915. Previous operation, tonsillectomy three years previously. Four days before examination a growth on the left side of the roof of the mouth was noticed; a physician was consulted, who punctured it, but did not take a specimen for diagnosis. He advised her to come immediately for treatment. It was not known how long the growth had been present; the girl believed that the left side of the roof of the mouth had always been larger than the right. There had been no other complaint; her general health was good, though she was very nervous. A tumor was found on the left side of the hard palate, two by one and one-half inches, extending into the soft palate. It was fairly regular in outline, and the mucous membrane covering it was normal except where it had been punctured. About this opening were signs of inflammation. The nose and sinuses were negative, and no glands were palpable in the neck. Wassermann negative. When taking a specimen from the tumor for diagnosis, about an ounce of straw-colored sticky fluid was evacuated. Microscopic examination of the specimen showed a cyst adenoma. (Figures 1, 2, 3 and 4.)

On December 13, 1915, the tumor was removed under ether anesthesia. It was found limited to the soft tissues of the palate, and did not involve the bone, although an excavation was noticed where it had rested. While the growth was readily dissected out, there was no definite encapsulation. The wound healed nicely and there is no recurrence to date.

Case 2 (156,093).—L. M. P., male, aged sixty-four years. Examined March 29, 1916. The man came to the Clinic because of a growth on the left side of the palate, noticed one year previously. The tumor did not seem to be increasing in size, and he had no inconvenience from it. Two weeks previously he consulted a physician, who lanced the tumor twice, but nothing was evacuated. No further treatment given. Except for this growth the patient was well. When examined, a tumor one and one-half by one inch in size was seen on the left side of the palate at the juncture of the hard and soft palates. Mucous membrane normal over the tumor except where it had been incised by the home physician; tumor freely movable.

On March 3, 1916, the tumor was excised under local anesthesia. The growth was definitely encapsulated and easily shelled out. The wound healed readily. Microscopically the growth proved to be a peculiar epithelial tumor. (Figures 5, 6, 7, and 8.)

Adenomas of the palate may occur at any age. The youngest patient was a girl, aged sixteen years, reported in this paper, and the oldest a man aged sixty-five years, reported by Escat[2] in 1897. In the group of proved cases collected, including the one I have reported, were eight females and one male. The duration of the tumor may be from one year to fourteen years, but often the growth has been found accidentally on examining the mouth. Often in cases of the smaller tumors no symptoms are complained of, but the growth may increase to such size as to cause dyspnea and difficulty in swallowing. Dobson[3] reports a fibroma adenoma so large that it could not be removed between the teeth. The tumor may be situated in the hard or the soft palate or may involve both. Most frequently it is seen on the left side. It is not characteristic in appearance. The mucous membrane generally appears normal and unbroken, although Hutchison[4] reports two cases in which the surfaces were deeply ulcerated. In one case the ulceration resembled the crypts of a tonsil and contained cheesy material. In these two cases and Case 157,265 reported here, the tumors had diffuse margins with no definite capsule. In this respect they were quite different from those of other cases reported. Usually the growth is freely movable and rests in a saucer-like cavity in the bone like a dermoid. The tumor may be cystic, as that of Case 157,265, and the case reported by Marsden and White.[5] The outline and surface of the tumor are generally regular. No glandular enlargement was noted in any case reported. King,[6] however, reports a case in which the base of the tumor passed outward and backward behind the jaw and could be felt externally. In order to remove the tumor it was necessary to split the cheek. Dobson[3] reports a case in which the growth formed a swelling the size of a chestnut under the angle of the jaw. This swelling, however, we believe was an extension of the tumor rather than a gland.

HISTOGENESIS AND PATHOLOGY.

Paget[7] states that pathologically these tumors are embryonic in character and resemble closely tumors of the parotid which are manifestly embryonic. Morton[8] says the fact that in these tumors the same cells differentiate into gland epithelium and embryonic connective tissue cells of oval or spindle shape, proves the embryonic origin of the tumor from an early stage in the fetal development, and indicates a close analogy to the complex nature of the ordinary parotid tumor. MacCarty believes, however, that they are not of embryonic origin, but develop from the germinative layer of the epithelium of the palate. The normal basal layer of this epithelium may develop squamous cells or differentiate into glandular cells. Although these two tumors originate apparently in the same cell group, they are two distinct types. In the one the basal cells have grown down and differentiated into glandular cells, forming the picture seen in the true cyst adenoma. In the other the germinative layer of the epithelium has formed squamous cells and cell nests in its development. Areas are to be seen in which the differentiated cells are disappearing and others in which apparently they have dropped out entirely, the germinative layer alone remaining about the cavity. This theory as to the origin of these tumors seems by far the most plausible.

Most adenomas of the palate are encapsulated tumors, homogeneous on the surface and on section. Those reported by Marsden and White[5] and that in Case 157,265, however, were definitely cystic, containing fluid, According to Morton,[7] the characteristic features of an adenoma on microscopic examination are (1) cell nests, (2) adenoid tissue, (3) glandular structure, and (4) embryonic connective tissue. Hutchison[4] states that, in addition to these features, the tumors reported by him showed immense numbers of acini like those of the salivary gland and oral cavities filled with lymph cells. The latter possesses a regular fibrous wall similar to that of the lymphoid follicles of the tongue, tonsils, etc. It would seem that the term adenoma should be reserved for the type of tumor showing the characteristics of Case 157,265.

DIAGNOSIS AND TREATMENT.

In differentiating an adenoma from other types of tumors of the palate, it must be remembered that adenocarcinoma is not infrequently found in this region and also that sarcoma may be present. This must be taken into consideration particularly in the diagnosis of cases in which the tumor is found accidentally and the history necessary to eliminate malignancy is not obtainable. By far the safest plan is to excise a piece of tissue for examination. An operation may then be performed with a definite knowledge of the treatment indicated. When the tumors are small it is best to remove them under local anesthesia, as usually no difficulty is found in excising them. When large, however, such as those reported by Dobson[3] and King,[6] a more radical procedure may be required.

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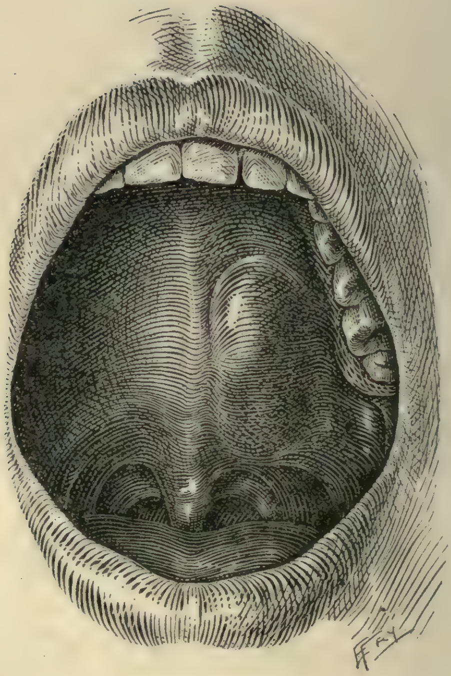

FIGURE 1. (157,265.)
Cyst adenoma of palate.

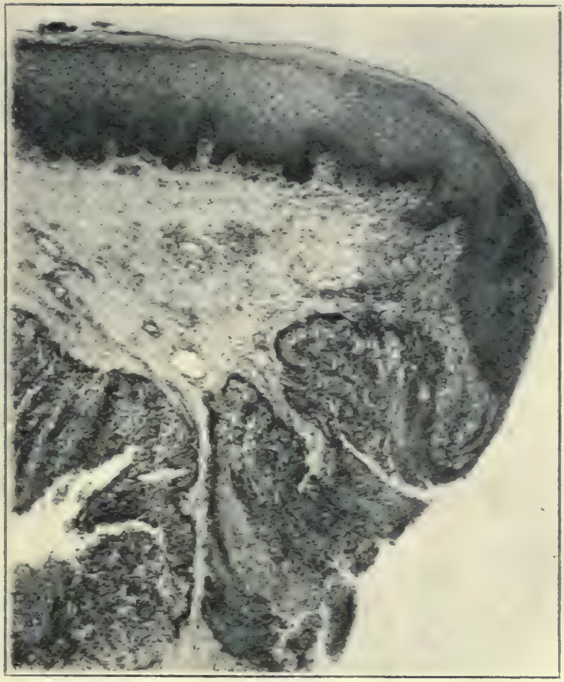

FIGURE 2. (Same as Figure 1.)
Downgrowth of the epithelium from the normal epithelium.

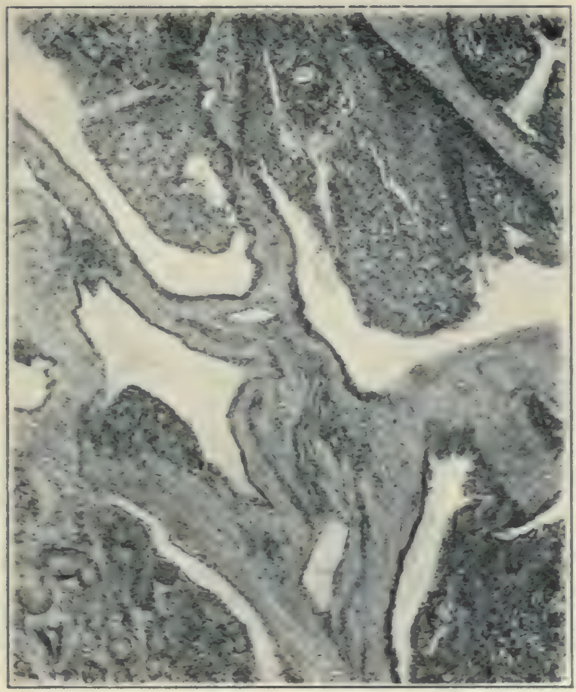

FIGURE 3. (Same as Figure 1.)
Glandular structure of the tumor.

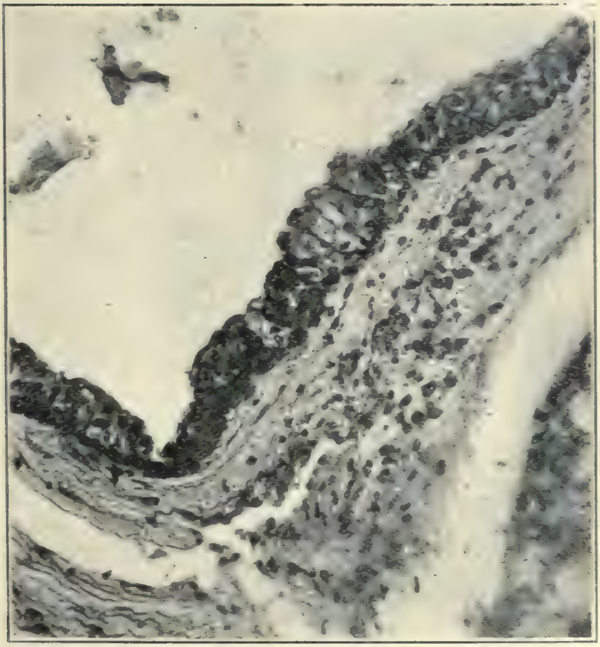

FIGURE 4. (Same as Figure 1.)
High magnification of the glandular cells.

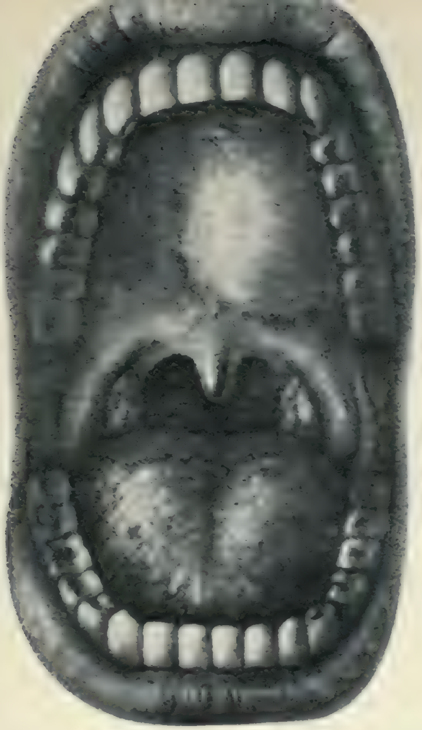

FIGURE 5. (156,093.)

Unusual epithelial tumor of the palate.

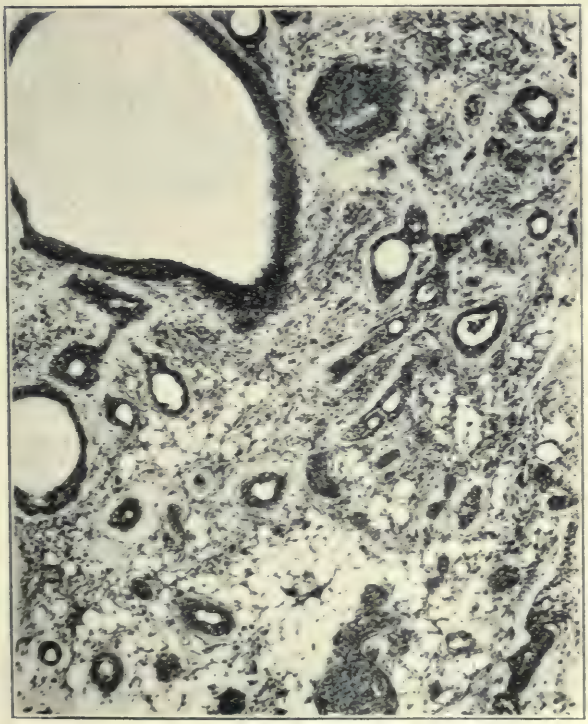

FIGURE 6. (Same as Figure 5.)

Areas showing the cells' nests. In other areas the central part of the nests has dropped out, leaving the outer layer of cells.

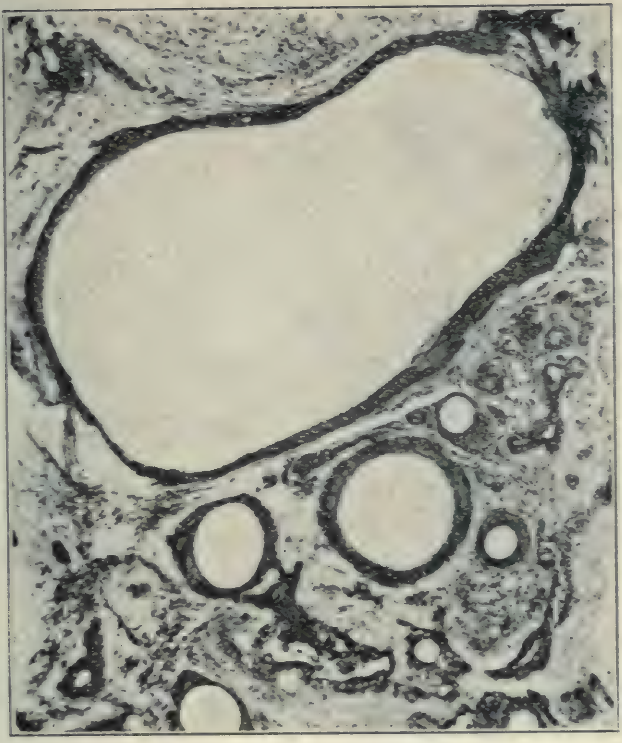

FIGURE 7. (Same as Figure 5.)

Several areas where the central portion of the cell nests has dropped out.

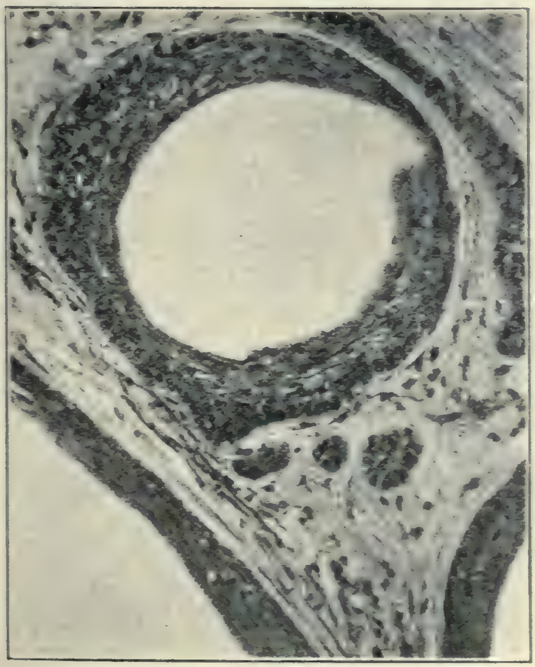

FIGURE 8. (Same as Figure 5.)

Character of epithelium in remains of cell nests (high magnification).

XLII.

SURGICAL ANATOMY OF THE FRONTAL SINUS. DEMONSTRATION OF ANATOMIC PREPARATIONS.*

By George E. Shambaugh, M. D.,

Chicago.

Although the frontal sinus, because of its location, is the most exposed of any of the nasal accessory sinuses, it is the most difficult to treat of all of the sinuses when it becomes the seat of infection. The exposed position of the frontal sinus renders it the most accessible for external operation, but it is the one sinus where the external operation is least permissible. For obvious reasons the external operation for the relief of frontal sinus disease is restricted to those exceptional cases where an external opening has already taken place, or where intranasal methods of operation have failed to relieve symptoms of persistent severe pain resulting from the pressure of confined pus. This restriction of the cases where the external operation is indicated has become more and more generally accepted as it has become apparent that by more careful, painstaking intranasal work fewer and fewer of the cases of frontal sinus disease remain which cannot be cured or at least satisfactorily relieved by intranasal surgery. The objectionable scar in the most conspicuous part of the face, which persists even after the most favorable external operation, should constitute a contraindication for this method of operation except as a measure of last resort for the cases just indicated.

There can be no question that the intranasal operation for securing adequate drainage of the frontal sinus is more difficult and more dangerous than is the case with the other accessory sinuses. The difficulty and the danger of the intranasal operation lies in the anatomic variations existing in different frontal sinuses and in the relations of the nasal opening of this sinus to the cribriform plate of the ethmoid. For these reasons an examination of the surgical relations of the frontal sinus is of more than ordinary interest and importance. Because of the great variation in the size and shape of the frontal sinus, it is not feasible to take any one preparation and describe it as the typical frontal sinus. In this demonstration I shall attempt to show the more usual variations in the anatomy of the frontal sinus.

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*Read before the Chicago Laryngological and Otological Society, February 29, 1916.

The first variation to which I shall refer is the absence of one or of both frontal sinuses. This is not an unusual variation. I have perhaps a dozen preparations showing an absence of one or of both frontal sinuses. This variation is, as a rule, bilateral. Occasionally there is a small frontal sinus on one side and an absence of a sinus on the opposite side. I have never encountered a large frontal sinus on one side with an absence of a sinus on the opposite side.

It is not always easy to make a distinction between a small frontal sinus and an anterior ethmoid cell, and such a distinction is often more or less arbitrary. The frontal, like the maxillary sinus, is an outgrowth from the ethmoid, and in a broad sense may be considered as an ethmoid cell. It is only when the cell is pushed upwards between the perpendicular plates of the frontal bone that the term frontal sinus seems applicable. Still, every ethmoid cell that has grown upwards between the plates of the frontal bone can hardly be called a frontal sinus. It is not at all uncommon for an anterior ethmoid cell to develop upward in this way within a well formed frontal sinus. Such a condition is not referred to as a double or a secondary frontal sinus. The ethmoid cell developing inside of a frontal sinus is still referred to as an ethmoid cell, under the name of bulla frontalis.

A well developed frontal sinus may exist where there is scarcely any extension upwards between the perpendicular plates of the frontal bone, but where the sinus lies entirely above the orbit.

In the most typical frontal sinus the development has taken place as an outgrowth from the anterior end of the infundibulum. In such cases the frontal sinus drains into the anterior end of the infundibulum. It is not at all unusual for the anterior end of the infundibulum to open into a small or large ethmoid cell, while the frontal sinus has developed as an outgrowth from that part of the ethmoid just mesial to the infundibulum. It is seen, therefore, that a distinction between the frontal sinus and an ethmoid cell can be based neither upon the location nor yet upon the opening into the nose. The distinction is often quite arbitrary.

Contrasting with these preparations showing a diminutive frontal sinus or a complete absence of the sinus, are the preparations showing an unusually large sinus. Such sinuses are of special practical interest for two reasons. They are especially likely to be the seat of severe symptoms when they become infected, and should an external operation be resorted to they are more certain to cause a very disfiguring scar.

In expanding the frontal sinus may enlarge in three directions: laterally until it reaches the outer angle of the orbit, posteriorly over the roof of the orbit, extending even to the posterior part of the orbit, and upwards between the perpendicular plates of the frontal bone, extending in exceptional cases almost to the margin of the hair. An enlargement of the frontal sinus outward and upward can often be detected from the fullness above the eyes. In these cases the transillumination test often gives a very good idea of the anatomic condition, while the skiagraph gives a very accurate outline of the exact size of the sinus. The depth of the frontal sinus in its perpendicular enlargement is of special interest, since the degree of depression resulting from an external operation is largely dependent on this factor. This depth of the sinus is always greatest when the outlines of the sinus are expanded. I have preparations showing the separation of a full inch between the outer and inner tables of the frontal bone near the floor of a large frontal sinus.

Partial partitions within the frontal sinus often exist. They usually extend downward from the upper margin, thus separating the upper part of the sinus into several pockets. In other preparations such partition plates are found dividing the horizontal extension of the sinus laterally above the brow, as well as posteriorly above the orbit into several chambers. These partition plates are placed so as to interfere very little, if any, with the drainage through the natural opening of the sinus, but their presence may interfere materially with the thorough exenteration of pathologic conditions within the sinus, even when performing the external operation.

The relations of the normal opening of the frontal sinus are of extreme interest in connection with the intranasal operation. They are by no means constant, but vary widely in different cases. As an unusual anatomic variation I have a single preparation in which the two frontal sinuses open into the nose through a single nasofrontal duct. The frontal sinus on the side where the nasofrontal duct is absent opens through a defect in the septum into the opposite frontal sinus. The one frontal sinus in this case has developed as an outgrowth from the other frontal sinus. The relations which are of particular interest are, first, those about the nasal opening of the nasofrontal duct; second, those of the ostium to the floor of the frontal sinus; and third, the relations of the ostium to the cribriform plate.

As regards the relations about the nasal opening of the ductus frontalis, it is important to keep in mind the fact that while the anterior end of the infundibulum opens typically into the nasofrontal duct, there are many cases where the opening lies to the mesial side of the infundibulum. In passing a canula into the frontal sinus it is sometimes necessary to introduce the point of the canula well back in the region of the bulla before it can be passed upwards and forced into the frontal sinus. In other cases the point of the canula must be placed far forward in order to allow it to pass into the sinus. It is sometimes necessary to use considerable pressure before a probe can be introduced into the frontal sinus. Such pressure is permissible only when the operator is perfectly familiar with the anatomy of this region and provided one adhere strictly to the following directions: The point of the probe must be kept away from the region of the cribriform plate—that is, it must be kept as far away from the septum as possible. The point must be directed more outward toward the orbit than inward. The point of the probe should be bent forward in a long curve, and should be directed at a right angle to the handle of the probe. This is necessary in order that when making pressure it will be possible to force the point of the probe forward and outward and only slightly upward. By following these directions it will be possible even to force the point of the probe through the fragile walls of an ethmoid cell into the frontal sinus in exceptional cases where the probe cannot be made to enter through the natural opening.

The relation of the ostium to the floor of the sinus is best shown by making a horizontal section about the level of the supraorbital ridge. The ostium is found, as a rule, well back close under the internal plate of the frontal bone. In cases where there is a supraorbital extension of the frontal sinus, the ostium may be placed as far back as the middle of the cribriform plate. The relation of the ostium to the floor of the frontal sinus is determined largely by the surrounding ethmoid cells. For example, an ethmoid cell developing in front of the unciform process may extend upward in such a manner as to push the ostium well back. In exceptional cases only does an ethmoid cell develop in such a manner as to push the ostium away from the posterior wall of the sinus.

XLIII.

REPORT OF A CASE OF ACUTE MASTOIDITIS WITH INFLUENZAL MENINGITIS. TREATMENT BY OPERATION ON THE MASTOID AND ANTI-INFLUENZAL SERUM, FOLLOWED BY BRAIN ABSCESS — OPERATION — RECOVERY.

By Francis R. Packard, M. D.,

Philadelphia.

E. C., eleven years old, was taken ill on Wednesday, October 2, 1913, on which day she came home from school, having been exposed to a severe wetting in a rain storm. That night she had a chill, followed by fever. A few days later she developed a pain in her right ear, which was followed by vomiting and headache. I was called in to see her, in consultation, by Dr. Knipe, the family physician, on the 12th of October, ten days after her initial chill. She complained of severe headache. Her temperature was 102°, pulse 120, and she seemed very irritable and had some photophobia. I incised her right ear drum and got a great quantity of pus from the right middle ear.

The next day, October 13th, she was admitted to the Pennsylvania Hospital, and I opened her right mastoid. Before the operation she was seen by Dr. Arthur Newlin, who agreed with me that she was suffering from meningitis, with acute mastoiditis. The child lay in bed, on the left side, with head retracted, crying out sharply from time to time. The pupils were rather large, reacted to light, and were equal. Her heart and lungs were negative. Kernig's sign was present to a slight degree. Her blood count showed 26,000 leucocytes, ninety per cent of which were polymorphonuclears.

Upon opening the right mastoid, which I did under ether, the process was found to be extremely cellular, and the bone very soft in consistence, a small amount of pus in the antrum and in the cells towards the tip. Whilst she was under ether a lumbar puncture was made in the fourth interspace by Dr. Billings, and twenty cubic centimeters of spinal fluid, under great pressure, was removed. The patient reacted quietly after the operation. Owing to the appearance of the spinal fluid and the history of the case, it was decided to give the patient the antiinfluenzal serum of Dr. Flexner, of the Rockefeller Institute, of which Dr. Richardson had some in the Ayer Clinical Laboratory. This was done the same evening, and fifteen cubic centimeters of the spinal fluid were withdrawn and thirty cubic centimeters of the antiinfluenzal serum were injected under ethylchlorid anesthesia.

On October 14th the following report was received from Dr. Richardson of the Ayer Clinical Laboratory of the Pennsylvania Hospital:

"The spinal fluid presented the typical picture of that from a case of acute meningitis, being very turbid. Noguchi's butyric acid test was positive, and Fehling's copper solution was not reduced. Microscopic examination showed many pus cells, almost all of which were polynuclears. There were present some organisms which resembled B. influenzæ. Some of the sediment was cultured on human blood agar. After twenty-four hours there appeared several fine white colonies, made up of a small gram negative bacillus. These on further study proved to be B. influenzæ."

The same day, October 14th, another lumbar puncture was made by Dr. Billings under ethylchlorid anesthesia, and thirty cubic centimeters of spinal fluid were withdrawn and thirty cubic centimeters of the antiinfluenzal serum injected.

On October 15th the patient was very much more comfortable, still had some photophobia, and Kernig's sign was marked in the right leg, with much less rigidity of the neck, and the child was brighter. She was again given ethylchlorid and about thirty cubic centimeters of spinal fluid were withdrawn by lumbar puncture from the spinal cord, followed by the injection of thirty cubic centimeters of the antiinfluenzal serum.

On the 16th the child's condition was much the same, except that she was much more irritable. The lumbar puncture and the injection of the antiinfluenzal serum was repeated, but very little fluid was withdrawn from the spinal column, and it was practically clear. Thirty cubic centimeters of the antiinfluenzal serum were injected.

On October 17th the child's condition showed most distinct improvement. She was cheerful and bright, with no rigidity of the neck, and Kernig's sign had practically disappeared. There was still some twitching of the muscles in certain parts of the body during sleep. This was the date of the last injection that the child received.

Dr. Richardson furnished me with the accompanying laboratory note as to his findings throughout the period in which the child received injections of the antiinfluenzal serum.

"The further examination of fluid during treatment with the serum showed a gradually improving condition. There were no organisms found after the first specimen. The pus cells decreased in number, and the butyric acid and Fehling's tests later became negative. Several specimens of spinal fluid were examiner. The last showed an almost normal fluid with only slightly increased number of white cells. The butyric acid test showed only the smallest possible flocculent precipitate, while Fehling's solution was reduced."

Up to this date the pus from the ear discharge showed a pure culture of staphylococcus aureus.

From this date, October 17th, which was four days subsequent to the operation and to the first injection of the antiinfluenzal serum, the child's case followed an uneventful course, until October 29, 1913. Her mastoid wound had practically healed, there being only a small surface on the lower portion which had not closed over. There was a slight amount of serous discharge from the external auditory canal.

On October 29th the child developed a slight temperature (100°), accompanied by some rapidity of the pulse. She complained of headache, and seemed very irritable. She had no Kernig's sign and no rigidity of the neck. Two days later, November 1st, she had several vomiting spells of a projectile character, and also seemed somewhat drowsy. Dr. William T. Shoemaker examined the eye grounds, and reported that the right optic disc was very hazy. Dr. Robert G. Le Conte saw the child in consultation with Dr. Arthur Newlin and myself. It was decided that the symptoms were indicative of brain abscess, and on November 3, 1913, Dr. Le Conte and myself opened the cranium. On exposing the dura it was found to be very tense and bulging, and a large abscess cavity was found in the substance of the temporosphenoidal lobe. In the course of the operation the lateral sinus was accidentally punctured, but the hemorrhage was controlled without trouble. A rubber drainage tube was inserted into the wound, which was packed with plain gauze.

The subsequent course of the case affords nothing of any particular interest. Drainage of the wound was done away with on November 11th, eight days subsequent to the operation, and the patient was discharged, cured, from the hospital on December 7, 1913.

Dr. Richardson's report on the pus from the brain abscess showed a pure culture of streptococcus.

I have reported this case because it seems to me that the primary meningitis was undoubtedly influenzal and that the antiinfluenzal serum proved efficacious in its relief. The results following its administration were so striking that they seemed to be conclusive. I think it is also interesting to note that the laboratory report showed that the pus from the ear discharge was a pure culture of staphylococcus; the culture from the spinal cord showed the influenzal bacillus; and the pus from the brain abscess showed a pure culture of streptococcus.

XLIV.

SUPPURATION OF THE ACCESSORY NASAL SINUSES AS A POSSIBLE ETIOLOGIC FACTOR IN MULTIPLE SCLEROSIS.

By H. H. Stark, M. D.,

El Paso.

Having had occasion in the last year to look up the literature on blindness due to suppuration of the accessory nasal sinuses,[1] I was impressed with the similarity of the eye symptoms in this disease and those of multiple sclerosis, this being particularly true in those cases of sclerosis in which the diagnosis is made on the early eye symptoms, which may be the first and only symptoms of the disease in many instances.

This latter fact is emphasized by Bruns and Stoelting,[2] who, in the tabulation of their cases, show thirty-two per cent with ocular affections preceding the other symptoms for a variable length of time. This percentage, by different figuring, could be raised to fifty-eight per cent.

In the study of one case of sinus blindness this similarity is not so marked as where the symptoms are tabulated in the order of their frequency, as it is seldom we find all of them present in one case, diagnosis usually being made by the finding of one or two symptoms, and in some cases the presence of only one.

The time allowed for my paper was too short to go into this subject, and I only mentioned it in my closing discussion, in which I said: "I was waiting for someone to bring up the question of multiple sclerosis. The eye symptoms of this disease are so much like the cases I have reported, that I feel it quite possible for the nasal sinus suppuration to be an etiologic factor in sclerosis of the brain and spinal cord." So far as I can discover, this was the first time this question was mentioned in literature. Since that time I have made a fairly good search of the literature, and have found many points to strengthen my theory that not only are the symptoms alike, but the two conditions may be due to the same cause.

I, therefore, think that a paper on this subject may be acceptable to the profession, and may do good by stimulating investigation, as my future work on this line will necessarily be slow, on account of the lack of neurologic material.

The eye symptoms in multiple sclerosis are amblyopia, nerve involvement, muscle involvement, including nystagmus, change of the pupil and field of vision. As in sinus blindness, these symptoms are not all present in one case, the diagnosis frequently being made by the presence of one or more, and it is necessary in certain cases to exclude hysteric amblyopia in the diagnosis of both diseases, but this can usually be done by the history of the case, interlacing peripheral color fields, and other signs.

I will take up the eye symptoms in the order of their importance and frequency, so that the similarity of the two diseases can better be seen.

AMBLYOPIA.

Ramer[3] classes sudden blindness as the most important eye symptom of multiple sclerosis. It is of a type resembling retrobulbar neuritis, and all authors agree that it may be the first and only symptom for a number of years, and it is usually monocular.

Aurbach and Brandt[4] state that in eighty per cent of the cases the blindness is monocular and comes without prodroma, or after transitory headache or vertigo. They say that the general symptoms come on as late as fifteen years after the blindness, as the disease is frequently very slow in its development.

Bramwell[5] says that visual defects, which are frequent and characteristic, often appear at an early date. Temporary unilateral amblyopia, of sudden onset and often regarded as hysteric in origin, as it is unaccompanied by change in the fundus, may be the first symptom of the disease. Other authors agree that it may be the first symptom, and that it may be a transitory blindness.

These symptoms just described agree in every respect to the amblyopia of sinus suppuration. The sudden onset, without prodroma, possibly a headache or dizziness, in many cases a normal fundus, and even the percentage of monocular cases is exactly the same, eighty per cent.

This may also be transitory, as had been shown by numerous authors. I recently reported a case[1] of a physician, whose onset was sudden, the patient being immediately relieved by a spontaneous discharge of a small amount of pus from the nose, on the affected side. One can readily see that with an ordinary patient this fact could be easily overlooked, had the attention of the patient not been called to it.

OPTIC NERVE.

Ramer[3] classes the involvement of the optic nerve as second in importance of the eye symptoms of multiple sclerosis.

Bramwell,[5] in speaking of the nerve changes, says there may be a pallor of the temporal side of the disc, or an incomplete pallor of the whole disc, a small number of cases having been observed with optic neuritis, never, in his experience, amounting to true papillitis.

Bruns and Stoelting[2] quoted Oppenheimer as having had a case of atrophy of the optic nerve, this being the only cerebral symptom for twenty years.

Aurbach and Brandt[4] say that the eye only a few times shows the presence of neuritis or papillitis, atrophy occurring in twelve per cent of the cases.

Optic nerve findings in sinus blindness can be any change from paleness of the temporal side of the disc, neuritis, even papillitis. Great emphasis is placed on the paleness of the temporal side by many authors, but we do not find the same percentage of change existing in the two diseases. In my compilation of eighty-eight cases from the literature, there were fifty per cent showing optic neuritis, ten per cent of which were to a degree amounting to choked disc, but the atrophy percentage is practically the same as in multiple sclerosis, being eleven per cent.

OCULAR MUSCLES.

Ramer[3] classes this as third in importance of the eye symptoms.

Bramwell,[5] in speaking of multiple sclerosis, says diplopia, with no visible impairment of the ocular movement, is an early symptom for which the patient seeks advice, and was present in thirteen of thirty-one cases recently examined by him, showing an involvement in his cases of forty per cent. He says that this involvement is of so slight a degree as to escape detection, unless specifically looked for.

Uhthoff[5] says twenty per cent of the cases have muscular involvement.

Ramer[3] says the muscular disturbance consists of (1) paresis, (2) nystagmus, or twitching of the eye resembling nystagmus. He says they are transient, often passing quickly, seldom lasting.

In my tabulation of sinus blindness there was thirteen per cent of muscular involvement, most of these being paresis of a transitory nature, as they were relieved by operation or spontaneous evacuation of the affected sinus; but if these symptoms are the same as multiple sclerosis, it must be especially looked for. There may have been a great many more with muscular disturbances which were never detected. So far, I find no case of nystagmus reported in sinus blindness, however, this might be present through toxic irritation of the labyrinth, which may also be the cause in multiple sclerosis.

THE PUPIL.

Ramer[3] classes this as fourth in importance of the eye symptoms in multiple sclerosis. He says that complete reflex immobility, miosis of the pupil, occurs very seldom, with occasional mydriasis and somewhat reduced reaction to light and a difference in the size of the pupil is met with.

Change of the pupil in sinus blindness is a point on which stress has not been laid. Two of my cases[1] had sudden dilatation of the pupil, occurring with amblyopia, but the pupil may be normal in size and reaction. In my tabulation of sinus blindness, I find reported twenty per cent with dilated pupil, four and one-half per cent with contracted pupil.

FIELD OF VISION.

Bramwell[5] says that in multiple sclerosis a central scotoma —usually bilateral—which may be for color alone, is often found, if looked for. He quotes Uhthoff, who classifies six different kinds of visual field: (1) Fifty per cent of the cases had central scotoma, usually bilateral and relative, with no change in the periphery. (2) Rarely peripheral contraction with scotoma. (3) A considerable percentage with irregular contraction. (4) Isolated cases with regular concentric contraction. (5) One case with ring scotoma. (6) One case with central scotoma, which cleared up and was followed by a contracted field.

Aurbach and Brandt[4] emphasize the importance of central scotoma for red and green.

In sinus blindness we have had reported fields of all classes mentioned. Early claims were that there was a peripheral contraction. Later emphasis was placed on central scotoma. In late years considerable attention has been paid to the enlargement of the blind spot. However, the latter is still a debatable question among the profession.

The majority of authors emphasize the importance of central color scotoma as a diagnostic point, Fuchs[6] reporting a case of sinus blindness with operation and recovery, in which diagnosis was made on the finding of a central color scotoma alone.

Birch-Hirschfeld[7] claims that the first symptom is central scotoma, which may be followed later by a peripheral contraction of the field.

My experience has been that the central scotoma for red and green is the most important; however, I believe that the field of vision in many cases depends on the anatomic construction of the orbit, the different fields being due to the difference in the applied pressure.

EXOPHTHALMOS.

So far, exophthalmos seems to be a symptom not considered in multiple sclerosis, but from the pathology there is no reason why it should not be present.

In sinus blindness it is mentioned in twenty per cent of the cases, but this no doubt was not all of the cases with this condition, as Elschnig[8] claims that it can be found in half of the cases, by examining with a Hertl exophthalmometer. So it is quite possible that this condition may have been overlooked in multiple sclerosis, as it certainly has in numerous cases of sinus blindness.

NERVOUS SYMPTOMS.

Aurbach and Brandt,[4] in speaking of the cases of multiple sclerosis beginning with eye symptoms, say that the nervous change is usually very slight, and must be looked for. They mention, among the many nervous symptoms, different sensations in the two halves of the face, and parethesis of the hands. Both of these symptoms are not infrequently found in known cases of sinus involvement, and no doubt these and other symptoms have been overlooked many times by the rhinologist, due to lack of careful investigation.

MENTAL SYMPTOMS.

Bramwell[5] says profound psychic alterations are exceptional. Even in fully developed cases there may be no mental disturbance, and he mentions emotional control being impaired. This condition is also given as one of the symptoms of pseudosclerosis.

In investigating this subject I was impressed with this point, as one of my cases had a symptom that was entirely overlooked in my previous history of the case. While operating on him the man laughed continuously. No significance was placed on this, but recently he has informed me that several times following the operation this has occurred. At one time he was awakened from a sound sleep, and continued laughing for as much as a half hour afterwards, the attacks coming on without assignable cause. It is true that this case may not be considered as one of uncomplicated sinus blindness, but clinically it worked out as such.

ETIOLOGY.

Bramwell[5] says the cause is most obscure. No factor common to a large proportion of the cases has been demonstrated. In a study of the different authors, one finds that almost every known cause has been suggested for multiple sclerosis, but with all authors there seems to run a line pointing to the possibility of infection as the causative factor.

Pierre Marie[9] says that sclerotic patches are due to medullary localization of general infection, basing his views on the clinical dependence of multiple sclerosis on infectious diseases.

Oppenheimer[10] says that with the majority of the cases the disease originates in an unknown base, or is a sequel after injurious factors, such as infection, and reports six cases following influenza. Competent neurologists are agreed as to the occasional occurrence of the disease after exposure to severe cold, especially in connection with abrupt changes of temperature.

Bramwell,[5] in considering the infection theory, says that the cases which seem to follow infections have no doubt had other symptoms present before the occurrence of the infection, infection simply making them worse.

Schnitzler[12] observed a case of multiple sclerosis in a woman of fifty-three years, following one month after an attack of influenza.

Church and Peterson[13] say the most important etiologic factors are infections.

Aurbach and Brandt[4] say that the relation of multiple sclerosis to retrobulbar neuritis is interesting in so far as the last named affection, in the great majority of cases, in acute as well as in the chronic form, is etiologically referrable to various intoxications and infections, arousing the suspicion that multiple sclerosis is likewise based on some organized or unorganized poison, crediting the increased frequency of influenza in the last decade with the greater frequency of multiple sclerosis.

If we admit that infection has something to do with multiple sclerosis (and we seem to have sufficient evidence in the literature to warrant this belief), we have exactly the same factor that causes sinus blindness.

PATHOLOGY.

In multiple sclerosis we find postmortem sclerotic patches in the brain and nerves. Just how early in the disease these sclerotic patches occur we are unable to say. However, it would look as though the process was slow, as is illustrated by Oppenheimer's case,[10] in which the general symptoms were delayed for twenty years.

Ramer[8] says that it has not been decided that the change of the nerve element or those of connective tissues and of the blood vessels are primary in the disease. He is inclined to the idea that the disease starts from the surrounding tissues and not from the nerve fibers, for the fact is established that the axis cylinders are spared for a long time in the foci of sclerosis in the optic sheath, and lose only their medullary sheath.

Parsons[11] says: "The pathologic anatomy of the optic nerve in insular sclerosis is characterized by a significant difference in detail from that in other diseases, differing in accord with those found in similar lesions in other parts of the central nervous system. It resembles tabetic atrophy in the absence of pronounced signs of inflammation; it differs from it in the prolonged escape in the great part of the axis cylinders, in spite of their denudation by destruction of the medullary sheath."

Of the vessels he says: "The blood vessels are greatly altered, often profoundly. When this condition is present, there is usually an increase in number and a widening of the lumen of the finer ones."

He also says: "The pathologic condition is suggestive of the presence of a circulating toxin as the cause of the disease, but there is at present no proof that such is the case."

It does not seem possible that transitory blindness, muscular, pupil and nerve involvement, could come from plaques found in the nerve, for if they did, these conditions would be permanent rather than temporary; so these plaques must be late in forming, making quite tenable Ramer's idea that the beginning is from without. The late formation of the plaques, no doubt, causes the permanent condition in the central nervous system.

The pathology of sinus blindness has not been thoroughly investigated, but we are sure that the trouble in the nerve comes from without, for it comes from the involved sinuses. This would give us the same condition found in the pathology of multiple sclerosis.

The process of sinus blindness is due, no doubt, to two factors, both causing pressure. First, a low grade cellulitis, set up by changes taking place in the orbit, causing a gradual pressure. Second, a sudden increased pressure in the orbit, caused by either partial or entire giving way of the bony wall, bringing sudden and direct pressure on the contents of the orbit.

When we consider that in these cases of sinus blindness, most of them have a closed or partially closed cavity, with no drainage through the natural openings into the nose, and realize that we have a condition of pus under pressure, which acts by a dialysis of the toxin, or, in certain instances, direct infection by the microorganism, we can readily see how this persistent poison will eventually change both the brain and the nerves. This would make Parson's theory[11] of a toxin in the blood of special significance. It is not necessary for this pus to be present in large quantities, as in a review of the literature on sinus blindness we find quite a number of cases where the contents of the affected sinus was very slight. The trouble is evidently due to a gradual absorption of the poison. The condition may have been latent for years and suddenly lighted up by an acute infection or other conditions. If our theory is true, this would explain Bramwell's idea that the condition had been present before the infection.

CONCLUSIONS.

Since starting the work on this paper a number of months ago, two cases have come to my notice which bear directly on this subject.

In July, 1914, while in correspondence with Dr. Frank Billings, of Chicago, he gave me the history of a case which was worked out by E. C. Rosenow. This case is not reported in the literature. He says: "During the last summer we had one patient in the Presbyterian Hospital who suffered from all the symptoms and signs of insular sclerosis, and showed infection about his throat. The tonsils were removed. From the cultures made from the tissues of the tonsils, streptococci were found which when injected into two dogs produced focal infection in the spinal cord and all of the symptoms of multiple sclerosis. After the removal of the boy's tonsils we have had word that he has improved very rapidly."

Recently E. A. Shumway[14] has reported a case of multiple sclerosis which is of great interest on account of its having gone through such competent hands. The patient noticed dimness of vision in one eye in 1907, at that time having vision of 6/22. This was diagnosed as sinus blindness due to suppuration of the posterior ethmoid cells on that side. Later vision fell to 2/45. After operation on the nose, vision gradually improved to 6/12, but again fell to 6/22, where it has remained. At the end of a year he noticed weakness of the right hand. From that time on varying symptoms of disease of the central nervous system developed, the diagnosis of multiple sclerosis finally being rendered in 1914, being agreed to by several neurologists who had examined him.

Shumway[14] says the case could either have been produced by sinus empyema or have been the initial symptom of the later developing of insular sclerosis, but from the light of subsequent events, it seems more reasonable to assume that the central nervous disease was the more probable cause. However, in his conclusion he says: "The query arises in my mind whether the disease in the present instance might have been caused by absorption of toxin from the purulent foci in the ethmoidal cells. So far as I know, no such theory has ever been advanced, and I offer it with some hesitancy."

These two cases strengthen my belief that multiple sclerosis is due to focal infection, and if statistics as given are, correct, that fifty per cent of the cases of this disease have eye symptoms, the natural supposition would be that these cases have their origin in the nasal sinuses. Possibly others without eye symptoms may have a like origin, but no doubt most of them came from other sources.

The eye symptoms of multiple sclerosis and sinus blindness, which have previously been considered different diseases, are to my mind the same, one being an advanced stage of the other.

Having no facilities for carrying on elaborate laboratory experiments, I leave this question to the future investigation of the profession, to either prove or disprove.

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SOCIETY PROCEEDINGS.

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLOGY.

Meeting of February 11, 1916.

Otitic Meningitis Complicating Scarlet Fever.

By Henry L. Lynah, M. D.

DISCUSSION.

Dr. H. L. Abramson: The meningitis department of the Bureau of Laboratories of the Department of Health was called in on the case March 20, 1915. We had received twenty cubic centimeters of turbid cerebrospinal fluid from the house physician of Willard Parker Hospital. Examination of this fluid brought out the following: A great increase in the cellular elements, ninety-five per cent being polymorphonuclears; organisms that looked like rather large Gram positive diplococci, and a Gram negative diplobacillus resembling the Friedländer bacillus; albumin, 3 plus; globulin, 3 plus; prompt reduction of Fehling's. We considered the organisms present in this fluid contaminations, due perhaps to unclean receptacle.

On first examination the child appeared to be in rather light stupor; slight anterior posterior rigidity of neck; a positive Macewen; pupils reacted promptly to light and accommodation; there were profuse purulent discharges from both ears; the knee jerks were exaggerated; no paralyses observed; no Brudzinski or contralateral; signs of involvement of right mastoid; temperature, 104 degrees; pulse, 160; respiration, 40. A lumbar puncture was done that afternoon and fifty cubic centimeters of turbid fluid removed under considerable pressure. Examination of this fluid showed the identical findings of the previous fluid. On the possibility that what looked like a Gram positive diplococcus in smear may prove to be a streptococcus in culture, twenty-five cubic centimeters of antistreptococcus was inoculated intraspinally.

Cultures from both fluids showed a mixture of a Gram positive diphtheroid bacillus and a Gram negative diplobacillus. These two organisms were separated, the former proving out as a chromogenic diphtheroid bacillus, producing yellow brown pigment, and the latter as a Gram negative diplobacillus, resembling the Friedländer bacillus. The diphtheroid grew very rapidly on ordinary nutrient agar, but the Gram negative diplobacillus grew scantily on plain nutrient agar but more profusely on glucose ascitic agar. The latter organism produced moist, mucoid, grayish colonies. Indol was produced in peptone solution, and it fermented lactose and dextrose. However, we were never able to demonstrate a capsule, even through cultivation in a fluid serum media.

On March 21, 1915, patient seemed brighter. Had been operated upon by Dr. Lynah for the mastoiditis of the right side the day before. He evacuated considerable tenacious purulent material. Lumbar puncture was done and fifty cubic centimeters of slightly blood tinged fluid was withdrawn. The fluid was thoroughly mixed with the blood, and it is our opinion that the blood was derived from the operation wound. This would indicate that there was free access between the seat of the inflammation in the mastoid and the subdural space. Twenty cubic centimeters of antistreptococcus serum was again inoculated. This spinal fluid and all subsequent specimens were free of organisms, both in smear and culture. Ptosis of upper eyelid and dilatation and immobility of the pupil on the left side were observed.

Lumbar puncture was done on March 22, 1915, and fifty cubic centimeters of straw colored, slightly turbid fluid was withdrawn under increased pressure. The color was due to hemaglobin derived from broken down red blood cells. The examination showed diminished number of cells, ninety per cent of which were polynuclears. Smear and culture were negative for bacteria; the content of albumin and globulin was diminished to 2 plus each; Fehling's solution was reduced promptly. The patient appeared to be brighter mentally.

Lumbar punctures were done on March 24th, 25th and 27th. None of these showed presence of organisms either in smear or culture. The cells, albumin and globulin content diminished rapidly, these elements in the fluid of March 27th being as follows: No increase in cells; albumin, 1 plus; globulin, a trace.

An autogenous vaccine of the two organisms was prepared and administered as follows: March 24th, 250 million; March 26th, 500 million; March 28th, 500 million; March 30th, 500 million.

Comment.—It is our opinion that the meningitis in this instance was localized to the comparatively small area of meninges adjacent to the inflammatory process in the mastoid, and, further, that the virulence of the organisms recovered from the spinal fluid was low grade.

That the process was localized is indicated first and foremost by the fact that the Fehling solution was reduced as in a normal fluid—that is, promptly from first to last. Had the process become generalized, it would have involved the sugar secreting mechanism, the choroid plexus, and by impairing its secretory function, would have caused a diminution or absence of the reducing substance in the fluid. The rapid decrease of the cells and albumin and globulin, too, points in the direction of the localization of the meningitis, for in the generalized meningococcic meningitis we find considerable increase in these elements for some time after the organisms have disappeared from the fluid.

The organisms were of low virulence and probably derived from the middle ear inflammation, as they are commonly found in purulent discharges from the ear. Their absence from the fluid after the operation was performed on the mastoid indicates that the readier drainage through the mastoid wound proved effective in clearing up this localized meningitis. Had the organisms been of a virulent character, there would no doubt have been an extension of the meningitis in spite of the operation.

The use of the antistreptococcus serum and autogenous vaccine deserves little or no credit for the favorable result of this case. The low virulence of the infection, the operation on the original focus and repeated lumbar punctures, account for the result.

Only one other case of meningitis due to the Friedländer bacillus has come to our attention. This was a case of cerebrospinal meningitis that had been treated with antimeningitis serum with favorable results. Not long after the spinal fluid had cleared up, he again developed symptoms of meningitis. Lumbar puncture brought forth a very tenacious mucoid exudate in very small amount, from which was recovered a pure culture of the Friedländer bacillus. On account of the character of the exudate and difficulty in removing same, a laminectomy was advised and performed. The patient drained more freely through this and made good recovery.

Diphtheroid bacilli are not very commonly found in the cerebrospinal fluid. Harris and Wade found them in three cases. In one case the smear was negative, but after two days' growth, culture showed a Gram positive diphtheroid bacillus. In the second case, a purulent meningitis resulting from a staphylococcus infected spinabifida, presented a few colonies of a Gram positive diphtheroid bacillus among numerous staphylococcus colonies, the diphtheroid being undoubtedly a skin contamination. The third fluid was from an adult male with high fever and opisthotonos. Forty cubic centimeters of slightly turbid fluid were withdrawn. This showed many pus cells and numerous Gram positive organisms, which proved to be diphtheroid bacilli. They were present in the specimen of fluid taken twenty-four hours later, but subsequently rapidly disappeared. Case recovered about a week after first lumbar puncture.

DR. DENCH said that the case was a particularly interesting one, as a meningitis of otitic origin is usually considered hopeless. With rare exceptions they are hopeless when operated upon, and it is advantageous to learn that certain cases do get well where the labyrinth is not extirpated or anything done except to relieve the pressure by lumbar puncture. It seemed to him that the bacteriologic findings and the vaccine therapy were rather mixed, as if the bacteriologist was harking back to the old shotgun prescriptions of thirty or forty years ago. We are still rather hazy as to the value of both the specific sera and the vaccines in cases of this kind. As he understood it, in this case the vaccines had been prepared from cultures from the spinal fluid. The case got well; that was the chief point. That is what all want to achieve, and the medication was certainly successful in this instance. By collating a large number of cases of this kind, we may be able to arrive at a definite conclusion as to what will be of value. We have some cases with a turbid cerebrospinal fluid and streptococci in the fluid, and on culture we find the organisms dead. The presence of any germ in the cerebrospinal fluid does not necessarily mean that it was the cause of the meningitis. That was why he had asked about the cultures, and whether they were made from a germ from the spinal fluid. In some way the cerebrospinal fluid may in certain cases kill the germ—the same as in some cases of brain abscess we find a sterile pus. We know that in certain of these cases of meningeal inflammation the otitic symptoms may be simply coincident. In other cases of undoubted otitic origin, the infective organism may die spontaneously and the cases recover. Only by collating a large number of these cases can any definite conclusions be reached. The report of Dr. Lynah's case was of great value on that account.

Patient Presented Five Years After Operation for Congenital Bilateral Microtia.

DR. JOHN RANDOLPH PAGE: The patient, a little boy, was only six weeks old when he first came under observation. Dr. Page watched the child carefully for two years before operating on the right ear. In studying the literature of the subject he had not found much encouragement to operate, three reasons for not operating being repeatedly advanced: First, that whenever there is a deformity of the auricle with atresia of the canal there is always coexistent such deformity of the labyrinth that hearing cannot be obtained. Second, that tuning fork and bone conduction tests are necessary to demonstrate hearing. Third, that it is impossible to secure an open meatus because of the cicatricial contraction which takes place after operation.

When this child was a baby, the mother was satisfied that he had some hearing. When he was two years old one could attract his attention by whistling on fingers. That was enough to demonstrate hearing, without tuning forks or bone conduction tests. On looking up the development of the ear, it was found that the essential organ of hearing develops from an entirely different origin from that of the auricle and external auditory canal.

The case was presented to show the meatuses. By examining the child it could be demonstrated that he did have considerable hearing. The mother claimed that she could call him from one room to another, without speaking louder to him than to her other children. In operating, allowance was made for cicatricial contraction. On one side the meatus now easily admits a speculum of medium size, and on the other side the largest speculum can be introduced. The first operation was performed five years ago, and the second was done four years ago.

One of the first things done after the operation was to test the labyrinth with water, to see if the child had a positive caloric, and the reaction was almost immediate.

When presented, the cavities were well dermatized, though there was some moisture in one. Dr. Page said that he did not remember in which one the tube was closed.

The mother stated that the child had called her attention to some frogs that were croaking a long distance off, a few months after the last operation.

This case was reported with a rather full account of the literature on the subject before the American Otological Society in 1914. In this report an interesting conclusion of Alexander's was mentioned, reached after many autospies on cases of congenital deformities of the auditory tract—namely, that in the vast majority of such cases the deformity is limited to the conducting apparatus alone, or to the perceiving apparatus alone; practically never is the defect present in both at the same time. He makes exceptions only in cases of nonviable embryos in which deformities of all three divisions of the ear have been found, and says that in such cases the deformity is always associated with other grave abnormalities elsewhere in the head, heart, or vascular system.

Another point of surgical value mentioned is the interesting observation accredited to Alexander by Castex and Burruyer, that malformation of the labyrinth always affects the facial nerve and auditory at the same time, and that one can conclude from this that all malformation of the external ear coexistent with facial paralysis indicates alteration in the labyrinth.

An X-ray was taken of this case when the child was small, and it demonstrated a tympanum; and at the operation rudimentary ossicles were found. Dr. Page said he believed that the stapes would be found in all of these cases.

DISCUSSION.

Dr. Braun congratulated Dr. Page upon the results obtained, and said that he would like to make a brief preliminary report and show some photographs of a case he himself had tested. Three years ago he had under his care a boy, five years of age, with a double congenital microtia. He had a slight amount of hearing if one shouted very loudly. He had not learned to speak. There was complete absence of the external auditory canal. There was a ridge which corresponded to the margin of the auricle. The left side was operated upon first, through an ordinary mastoid incision, which revealed a fairly normal mastoid. Dr. Braun said he was able to demonstrate a normal antrum, aditus, and attic. In the attic were seen a normal incus and malleus. The articulation of the jaw lay against the anterior surface of the mastoid. The tympanic cavity was normal, but there was no drum membrane. In place of the drum membrane there was a mass of fibrous tissue, against which the jaw lay. He did not do a radical because the middle ear was normal; but he opened up as widely as he could and left the ossicles in situ. A circular opening was made where the normal meatus should have been, and the small skin flap was turned into the cavity, and the mastoid incision sewed up. An effort was made to get the cavity to epidermatize, but without success, and after five or six weeks the cavity had almost completely filled up with granulations. It was curetted out, and a skin grafting was done. This was successful.

Some months later the second ear was operated upon, and the same conditions were found as in the left side.

The child has been going to a public school for a little over a year, has been advanced twice, and his teacher has no difficulty in making him hear. He hears a moderately loud conversational voice without any trouble.

Dr. Page said that he had been greatly interested in Dr. Braun's case, and with Dr. Braun's permission had included it with some others in the published report of his case. He had not followed the procedure of opening only down to the aditus, which was done as early as 1837 by Michael Jaeger, who advocated opening the antrum and allowing the sound waves to pass through the aditus to the tympanum without invasion of the latter. Dr. Duel, in the case of a girl ten or eleven years of age, had followed that method several years ago, and later placed her in Dr. Page's care for dressing, but it had not been possible for him to keep the opening patent, so in the case presented this evening he had decided to make as large an opening as possible, exposing the internal wall of the tympanum. In all of these cases the anterior wall of the bony canal is absent, and the articulation of the jaw encroaches on the mastoid and posterior canal wall. In Dr. Page's own case he had been able, by entering the mastoid far back, to make a large radical cavity. Alexander attributes the great number of failures in these operations to the fact that the operators do not realize the position of the jaw in these cases and try to open not through the mastoid, but in what they take to be the line of the external auditory canal, and consequently come into contact with the jaw, and possibly paralyze the facial nerve. Dr. Page said he believes that the safest procedure is to enter far back in the mastoid and expose the sinus and middle fossa; then having these landmarks, one can be guided accordingly and work from the aditus and horizontal semicircular canal outward and backward, and have the opening point inward and slightly forward to the tympanum.

Report of a Case of Serous Labyrinthitis.

DR. CHARLES E. PERKINS: Miss M. C., aged eighteen years, had a mastoid operation on her right ear six years ago. The posterior wound healed, but the ear continued to discharge, and for the last three years patient has complained of attacks of pain. Otoscopic examination late in July, 1915, showed the membrana tensa absent and the internal tympanic wall dermatized except over the posterior part in the region of the oval and round windows, where granulations in small amount were present. Granulation also protruded from beneath the posterior fold. Shrapnell's membrane was intact.

Functional tests showed hearing in the right ear for whisper, two feet; upper tone limit, 21,000; lower tone limit, 90 D. V. Left ear: low whisper, twelve feet; upper tone, 21,000; lower tone, 55 D. V. Bone conduction increased on right side. Caloric, positive on both sides. Rotation, positive—slightly less to the right side. No nystagmus, vertigo or nausea.

Radical operation was performed in Dr. Dench's service at

St. Luke's Hospital, with primary skin graft, on August 2, 1915. Vault and antrum filled with granulations which extended into the old mastoid cavity, which had partly reformed by bony growth from the periosteum.

The postoperative rise in temperature was not over 100°. Two days after operation there was slight vertigo; four days after, there was marked vertigo, nausea, and vomiting; nystagmus to the left—that is, to the healthy side.

The pledgets were removed from the wound. The graft was firmly adherent to the radical cavity except in the posterior part of the tympanum, where it was separated from the bone by the accumulation of blood. After removing the blood, the patient was able to hear the shouted voice. The tuning fork on the vertex was heard better in the unoperated ear.

The patient assumed the characteristic decubitus of destruction disharmony—that is, lay upon the side of the good ear, thus enabling her to look in the direction of the slow component of the nystagmus and minimize the vertigo. After two days the vertigo diminished rapidly and she was able to be up. Four days later, that is, eight days after the operation, examination showed hearing for shouted voice; caloric reaction sluggish, but positive; rotation produced nystagmus to the involved side for about twelve seconds. The patient continued to become dizzy upon stooping or turning around suddenly for about two months; but the nystagmus to the left, that is, toward the good ear, continued with scarcely noticeable diminution for five months, since which time it has been gradually disappearing.

The hearing in the right ear has returned to about the same as before the operation; the caloric reaction is positive; rotation produces a nystagmus to the right of about fifteen seconds, although the eye movements are sluggish.

In my experience it is unusual for nystagmus to persist in a marked degree for five months, after either a serous or a purulent labyrinthitis. If the static function of one labyrinth is totally and permanently ablated, as in diffuse suppurative labyrinthitis, the opposite labyrinth accustoms itself to the new order of things, and the symptoms of destructive disharmony, including the nystagmus, pass away within a month, as a rule. In this patient, the fact that the function of the static mechanism on the right side has been gradually returning seems in some way to act in causing the nystagmus to the left to persist.

The process within the labyrinth was no doubt caused by inflammation or the accumulation of blood beneath the graft at the side of the oval and round windows. Labyrinthine complications are more common in patients who present granulations in the neighborhood of these fenestra; so before applying a graft in these cases one should be sure that the hemorrhage has ceased and the tympanum is dry.

Not infrequently there is nystagmus and vertigo during the first four days after the radical operation, and not due to any intralabyrinthine disease. One hesitates before removing the grafts in such cases, but the possibility that there may be some disease of the labyrinth makes one more or less uneasy.

It is a well known fact that deafness which gives tests characteristic of lesions of the perceptive mechanism is usually an early symptom in serous and purulent meningitis. I have been accustomed to rely upon the Weber test in determining this point. For instance, if the fork placed upon the vertex is lateralized to the operated ear, it is, to a certain extent, an indication that the perceptive mechanism is intact. The patient must be fairly intelligent. There are, no doubt, possibilities of error in this test, but the information afforded has seemed to be of value.

DISCUSSION.

Dr. Braun asked if the fistula test was made before the operation, to which Dr. Perkins replied in the negative. Dr. Braun then said that in many of the cases in which a serous labyrinthitis followed the radical mastoid operation, a labyrinthine fistula was present before operation.

Dr. Braun said that he disagreed with Dr. Perkins as to the value of the Weber test. This had proved a very unreliable test in his hands. In a number of instances, patients with a dead labyrinth referred the Weber to the diseased side. In a patient operated on by Dr. Richards several years ago, in which he did a complete labyrinthectomy, the patient lateralized the Weber, after operation, to the ear from which the cochlea had been removed.

Dr. Friesner emphasized what Dr. Braun had said about the Weber test. If one has a suspected labyrinthitis, and the

Weber test be referred to that side, one would conclude naturally that that ear has function. He recalled a case upon which Dr. McCullagh had operated, in which the cholesteatoma had completely destroyed the labyrinth. The patient was a very intelligent young man, and he referred the Weber definitely to the diseased ear. Dr. Friesner was misled by this, and believed at first that the diseased ear had function, but the findings showed that the labyrinth was completely destroyed. Furthermore, the other tests showed that the labyrinth had no function.

Dr. Dench said that he also would like to emphasize this point in cases where there was not a destructive labyrinthitis, but where the labyrinth was involved from a nonsuppurative process. He had seen patients who were absolutely deaf in one ear refer the fork to the deaf side. He had seen that himself, though he did not know the cause, or how to explain it. It certainly does occur in suppurative labyrinthitis and in cases where the labyrinth has been removed. It occurs also in the nonsuppurative labyrinthine cases.

Dr. Perkins said that he was not unaware of the uncertainty of the Weber test, and had not claimed for it infallibility. In the presence of deafness due to lesion of the perceptive mechanism, we do not expect increased bone conduction. The fact that deafness of this kind is an early symptom in both serous and purulent meningitis, justifies us in expecting a diminished bone conduction in the involved ear. That there are exceptions, real or apparent, to this rule will not be denied. Nevertheless, he did not feel that he had claimed too much for this bone conduction test. No doubt, the safest way would be to remove the dressing and test the hearing, using the noise apparatus on the opposite side.

A Contribution to the Pathology of Mastoiditis With Reference to Thrombotic Conditions. Lantern Slide Demonstration.

Dr. Samuel J. Kopetzky said that he had not prepared a paper on this subject, but that he had been doing some work with the idea of coordinating the clinical findings with a study of the pathology of these cases, and that he presented these specimens at the invitation of the chairman.

DISCUSSION.

Dr. McCullagh said that this work which Dr. Kopetzky had been doing was most interesting and instructive. He had been especially interested in what had been said about the pathology of mastoiditis—the division of cases into the hemorrhagic and the coalescent types. He himself had felt that the latter was purely a secondary stage of the hemorrhagic condition which Dr. Kopetzky had described, and he believed that the hemorrhagic type does go on to this coalescent type in many cases. It would make the slides of more value if one could know the length of time that the disease had existed before the specimens were taken; it would give more light upon the time of change from the hemorrhagic to the coalescent type, if one merges into the other.

Paper: The Surgical Treatment of Persistent Suppuration of the Jugular Bulb.

By John McCoy, M. D.

DISCUSSION.

Dr. Dench said that the members of the section were indebted to Dr. McCoy for his clear exposition of the various operations for the relief of thrombosis involving the jugular bulb. They had been very skillfully collated and presented. There were good points in each operation. As Dr. McCoy had stated, the position of the jugular bulb varies in different subjects. In some cases it is the same on both sides; in some cases the dome is high on one side and low on the other; in some it is away forward, in others away back; and in some there is no dome. These latter cases are easy to operate upon.

Dr. Dench recalled two cases where he had done the Gruenert operation. Both patients died, but the operations were not particularly difficult. In both instances the jugular vein had been excised previously for a jugular sinus thrombosis. In all cases he has made the mastoid incision continuous with the incision in the neck, for the ligation of the jugular vein. In one instance the vein had been taken out by an assistant, who had not taken up the vein as high as he himself was accustomed to doing. It was a simple matter to pick up the end of the jugular vein in the neck in this case and follow it upward, then expose the sinus and work forward toward the bulb; then, by working far behind there was no danger of injuring the facial nerve. The facial nerve was cut in the first case. That was practically the only danger. Dr. Dench said that he almost always cuts the occipital artery in cases of jugular excision. He was in favor of a combination of the three methods in bulb cases. In the first place, make the mastoid incision continuous with the incision made in the neck for the jugular excision; then the vein and the nerve are exposed. It does not make much difference which technic is employed, for the technic adapted to one case might not be suitable to another, but by remembering the anatomic points the operation was freed of all difficulties.

All of his operations had been secondary to previous sinus and jugular operations. The primary cases must be very rare. He had followed one procedure in two or three cases. If one works very far forward and exposes the lateral sinus, in cases of sinus thrombosis, and follows on to the horizontal portion of the sinus to where the external wall becomes the inferior, it can be followed for a very considerable distance. In these three cases there was a considerable suppuration in the bulb, and it was easy to clean it out and pack it. The patients got well very rapidly. Dr. Dench said that where it was necessary to do the radical procedure he believed the best results could be obtained by a combination of the various methods presented by Dr. McCoy.

Dr. Whiting said that it has often occurred to him that the very elaborate methods of accomplishing results might be eliminated by doing a thorough operation in the beginning. In saying this he meant no reflection on the method of any operator, but he has performed a great many operations for sinus thrombosis and has never yet had a thrombosis of the bulb, although he has had a good many cases of thrombosis of the bulb in connection with operations by other men. As the sinus operation is commonly performed, the men content themselves with exposing an insufficient portion of the sinus. They expose the knee and the descending portion, and when they get fairly well down they speak of having carried the incision as far as the bulb. They are nowhere near the bulb, but only where the descending part of the sinus moves around in approaching that part.

In his own operations, Dr. Whiting has always followed the procedure mentioned by Dr. Dench, exposing posteriorly a good part of the occipital bone; then, instead of cutting the groove in the dimensions which correspond to the concavity, going posteriorly and cutting away all the jugular portion of the occipital bone between the transverse process. Then the operator has a space in which he can follow forward the sigmoid sinus into the bulb. That is a modification of Gruenert's procedure, except that it is not necessary to subsequently open the neck, as Gruenert does. Dr. Whiting said that he has done this many times in operating after other men, and has never had any trouble in cleaning out the bulb. Gruenert's operation is a well recognized one, but Dr. Whiting has never found it necessary to perform it.

Dr. Whiting is convinced that if the primary operation is performed, removing a large part of the occipital bone well back to the jugular process, then following the curve of the jugular sinus toward the jugular foramen, the posterior and lateral border can be taken away and the jugular border will be open, and it will never be necessary to do the Gruenert operation.

Dr. Braun said that he had occasion to open the jugular bulb a year ago, and was surprised to find how easily it was opened from behind, exposing the sinus well down (demonstration on blackboard), and with a rongeur removing the wedge of bone between the lower part of the vertical limb of the sinus and the bulb, keeping to the medial side of this wedge, in order to avoid the facial nerve. It did not take more than five minutes to make an opening half an inch in diameter. As there was a good deal of inflammation around the vein in the neck, the neck incision was extended up to meet the mastoid incision, and a probe passed from the open end of the vein up to the bulb. The case recovered.

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLOGY.

Meeting of March 10, 1916.

Entotic Aural Bruit, Audible on Auscultation of Head, Due to Tumor of the Pituitary Region.

Dr. L. D. Alexander: The patient, George D., was a negro, forty-two years of age, born in St. Kits, West Indies. He was married and had four children. Father was alive, eighty years of age; his mother died when about fifty (?); she had sixteen children.

Previous History.—Had measles at six years, no complications; had frequent headaches, right frontal and occipital regions, until twenty-one years of age. These disappeared when he left the West Indies. He denies venereal infection. Two years ago had a painful swelling of the left testicle, which subsided after rest and suspension. Has had hay fever every summer for the last fourteen years.

Present History.—Following his hay fever attack eighteen months ago, noticed a hissing sound in his right ear. This sound varies in intensity, and is especially noticeable when in a quiet place or upon exertion. He has had diplopia for the last year, and notices increasing difficulty of vision in the right eye; has daily headaches over the frontal and occipital areas, which are especially severe in the daytime.

Examination.—General physical condition, good. Post cervical glands, palpable. The chest and abdomen reflexes are negative.

The left ear is negative. Auscultation over the right ear reveals a blowing bruit, synchronous with pulse beats. The sound is heard directly over the entrance to the canal, and less distinctly forward and upward over the temporal region. The sound can be obliterated by firm pressure over the carotid.

The tubes are patent; tympanic membranes, normal; hearing, normal—Weber, Rinne, and forks.

Eyes are prominent. Pupils react to light and accommodation, but rather more slowly than normal. Media, clear; fundi, negative; optic disc and vessels appear normal; no tension. Vision: right, 20/30 plus C plus 3.50 sph.; left, 20/20 minus C plus 3.50 sph. Fields generally contracted for form and colors. No scotomata. Has diplopia increasing to left; image level; diplopia is light and indicates a slight paresis of left rectus; motility good.

Pathologic Examination.—Urine, negative. Blood, negative. Wassermann, 4 plus. Blood pressure, 140.

Radiographs of chest and anterior view of sinuses are negative. The lateral view shows enlargement of the sella, with probable erosion of some of its walls.

Radiographer's Report.—Plates 3743, December 20, 1915; 3743b, February 21, 1916. "The sella measures fourteen millimeters long by thirteen millimeters deep, and the area (averaged from the four plates by means of the planimeter) is one hundred and forty-five square millimeters, which is considerably larger than it should be. The posterior clinoids are elongated by apparent erosion at the base. A tumor in this location would be in contact or very close to the internal carotid as it leaves the carotid canal in the petrous pyramid; or there might be an aneurism of the carotid, possibly both tumor and aneurism, which would account for the bruit.

Sphenoidal sinuses are large and clear.

(Signed) GEORGE S. DIXON, M. D."

DISCUSSION.

DR. CHARLES GRAEF said that he was especially interested in this case, as he had been the first to see it, the patient having been referred to him last year by a practitioner for tinnitus and diplopia. He had made a diagnosis of aneurism, but had not determined the exact location of the vessel involved. It would seem to him that if the X-rays were to be accepted here as decisive, the pituitary gland should be expected to show more evidence of disturbance after a period of involvement lasting over a year, as this one has. No localizing signs or symptoms were found outside the X-ray shadows, and in intracranial conditions there were not very trustworthy. While under his care, the Wassermann reaction being four plus, the patient was kept on mixed treatment and given a series of mercurial inunctions. Outside of that, he could think of nothing likely to be of value but salvarsan, and as that was very expensive and difficult to obtain, the patient was not admitted to the hospital, and drifted into other hands.

He expressed his gratification at hearing the complete history given and at seeing the case here. He had never before heard of anything like this case. He had spoken of this patient to some of the members some weeks ago, and had intended to arrange for its presentation before the section.

DR. ALEXANDER in discussing the case stated that he had shown plates to Dr. Quimby, who agreed with findings of Dr. Dixon.

DR. EAGLETON said he believed the condition was due to an aneurism of the carotid which had widened the sella by pressure, and that he did not believe the pituitary involved.

Labyrinthitis Following Mumps.

DR. NORTON L. WILSON said that he presented this case not because of its rarity, but with the hope of either being able to do something for the restoration of the patient's hearing or to convince her that nothing could be done.

The patient, Marguerite F., was fifteen years of age, and had never had any trouble with her ears before the present attack. On December 25, 1915, she was taken ill with diphtheria; that same evening she was given four thousand units of antitoxin. The throat cleared up rapidly. On January 1st the left parotid gland became swollen, and the next day the right gland became involved. On January 5th, five days after the left gland became involved, she had some dizziness accompanied with nausea. On the 7th she had severe vertigo and vomiting.

She was seen by Dr. Wilson in consultation with her physician on the 8th of January, at which time she could not raise her head from the pillow without vomiting. The external canals and membrana tympani were normal. She was deaf in the left ear and complained of subjective noises; the noises still persist at times. The vomiting was not of the projectile type, and she had no fever at the time Dr. Wilson saw her. The vertigo lasted about two weeks, while the vomiting lasted only two days. At the time he saw her there was no nystagmus.

With the finger in the right ear she hears some sounds, but with the noise producer in the right ear she does not hear the loudest shout. The static labyrinth is functionating, as shown by the caloric test.

DISCUSSION.

Dr. Dench said that these cases are not so very common, and he would like to know from Dr. Wilson what had been done for the patient therapeutically. Sometimes pilocarpin helps these conditions. He had seen certain cases of affection of the cochlea following mumps in which it had had a very beneficial effect. Just why the vestibular portion clears up and the cochlea does not, he did not know. The cochlear labyrinth has rather better circulation from the anatomic point of view than the vestibular. The only reason he could think of was that the internal structures are rather more delicate in the cochlea, and while the gross circulation of the cochlea is better, the anastomosis in the semicircular canals is more free. Perhaps another reason is that though we are apt to consider the three semicircular canals as being on three exact mathematical planes, they are not so, and each semicircular canal takes up a part of the function of the others. We may for this reason have a functionating static labyrinth while the auditory labyrinth is destroyed. He strongly advised using pilocarpin in this case.

Dr. Blackwell, referring to independence of the cochlear and the vestibular branches, cited two cases which he had seen, in each of which the cochlea function was entirely lost without any history of vestibular irritation. Both presented perfectly normal vestibular action to both caloric and whirling tests.

Dr. Wilson, closing the discussion, said that he had nothing to add excepting to say that he was familiar with Dr. Dundas Grant's paper on the use of pilocarpin, but understood that he uses it in the earlier stages of the condition.

Lymphangiosarcoma of the Concha.

Dr. Hugh B. Blackwell: The patient, a boy, fourteen years of age, with a negative family history, was apparently in good health. The present trouble began eight months ago as a thin red line in the cochlea of the ear. This line gradually increased in size, raising and stretching the overlying integument, until nearly three months ago, when the skin covering became eroded and broke down; within a week the tumor was converted into a raw bleeding mass. During the following two weeks the patient had three hemorrhages from the tumor, one of which was quite severe, lasting for an hour and only controlled by continuous pressure.

The patient first came under Dr. Blackwell's observation nine weeks ago in the otologic clinic of Dr. Robert Lewis at the New York Eye and Ear Infirmary. At that time the tumor was about five times its present size, filling the lower portion of the concha. Its upper half was devoid of any skin covering and bled freely when touched. At first the appearance was somewhat that of an ordinary aural polyp. There was no glandular involvement.

Closer inspection revealed the fact that the growth was attached to the floor of the concha and that the external auditory canal and membrana tympani were normal. The tumor was cocainized and removed as closely as possible to its base by means of a wire snare. Its removal was followed by a very free arterial hemorrhage which came from a large vessel located in the center of the pedicle. This was controlled by means of a pressure bandage, and the tumor was sent to the laboratory for examination.

Laboratory Report.—"I beg leave to report in the case of Thomas McH. (Clinic No. 3402) that the growth removed from the concha is covered with epithelium having a slight tendency to digitate. The main portion of this tumor is composed of irregular connective tissue cells in which is found a very liberal supply of blood vessels or channels, some with no walls whatever and the balance lined with a single layer of endothelium. Some lymphoid cells being also present, the histologic picture of lymphangiosarcoma is completed. I would add that pedunculated sarcomas in this region are not likely to recur if thoroughly removed.

(Signed) George S. Dixon, M. D."

Conclusion.—The condition is very rare.. Politzer, in his book published in 1909, mentions only one case, that of Cohen Servaert. It is the first instance of its kind that I have observed in seven years' experience in two large active ear clinics. In connection with the previous history of hemorrhage, it is worthy of note that Jüngken states that in the simple angioma sometimes seen in this location a fatal issue may result from the rupture of vessels. On admission to the clinic, the interesting significance of the case lay in its close resemblance to an ordinary aural polyp—illustrating as it does the necessity of a careful differential examination in these cases. Of course, it is recognized that the histologic examination of a tumor may lead to a widely different appearance from one based on its clinical picture. In view of Dr. Dixon's careful examination and the clinical evidence submitted, Dr. Blackwell believes that despite its present innocent appearance the growth should be regarded as an early malignant recurrence and should be extirpated by means of what now would constitute a comparatively simple operation.

Acute Mastoiditis—Perisinus Abscess, Three Months' Duration—Scant Middle Ear Symptoms.

DR. JOSEPH COLEMAN: These two cases of mastoiditis which have come under my observation during the last few months belong to the class designated as atypical or insidious; atypical, in that the almost constantly existing symptoms which we are accustomed to find in acute mastoiditis, are either partially or totally absent; and insidious, because of the apparent slowness of the process, the coexisting extensive tissue destruction. I will report these two cases as briefly as possible.

Case 1.—Mr. P. M. B., aged forty-five years, came to my office December 22, 1915, complaining of inability to hear and pain on the left side, in the region of the mastoid antrum. From his history I learned that three months previously he had sudden severe pain in this ear, for which a paracentesis was done a few days later. Following the incision in the drum, he states that in spite of strict observation no discharge was seen. Examination of the hearing in the affected ear showed a watch which is normally heard at thirty inches to be heard at two inches, whispered voice at four feet, spoken voice at ten feet, Weber lateralizing to the left, the affected side, and Rinne diminished positive. The lowest fork heard was the 64, while all the high forks were heard. Upper tone limit normal. Examination of the ear showed the canal to be perfectly dry, drum intact, but slightly congested and thickened, resembling the condition found in a resolving acute catarrhal process. There was no bulging of the drum, and no sagging of the bony canal wall. Patient presented himself three days later with slight swelling behind the ear, and diminution in pain. He was sent to the New York Eye and Ear Infirmary and operated the following day. No X-ray was taken.

There was found a good sized perforation of the cortex. The cavity of the mastoid was full of pus and granulation. The cellular structure was broken down through almost the entire extent of the bone. The inner plate was eroded, exposing an area of sinus about three-quarters of an inch in length, bathed in pus, and covered with a heavy layer of granulation. The cerebellum was also exposed, with quite a large exposure in the middle cranial fossa. Bacteriologic examination of the smear taken from the mastoid at the time of operation showed long chain streptococcus. Discharged from hospital January 4, 1916.

Case 2.—Mr. J. B. J. presented himself with a history of having had pain in the right ear five months previous. Paracentesis had been performed, but no subsequent discharge was noted by the patient. He has now a large swelling behind the right ear, which has existed for two days. The canal is dry, the drum membrane intact, pearly white, of normal contour, and there exists no sagging of the bony canal wall. There is no pain. Case seemed to be one of local infection. Patient was advised to enter the infirmary. At the time of operation a probe was found to extend deeply into the wound, and disclosed an area of bare bone. Suspecting a fistulous opening, the incision was extended, and an enormous perforation in the cortex was found over the antrum. The sinus had been exposed by the process from the knee to the bend below, where it turns in toward the bulb. There was also an exposure of the cerebellum on each side of the sinus. The entire area was covered with pus and dense granulation.

In this case there existed no indication of infection of the middle ear, though account was taken of the fact that a paracentesis had been done five months before. The cells of the mastoid were obliterated and the bone sclerosed. In the region of the zygoma the bone was soft and necrotic. There was no X-ray taken or blood examination made. Smear taken from the mastoid at the operation showed streptococcus capsulatus.

In both of these cases the process seemed to have undergone resolution in the tympanic cavity, then going inward and eroding the inner plate, coming to the surface later. In the second case, resolution in the middle ear had been so complete as to leave no indication of the infection. Undoubtedly the route of infection in both these cases was through the middle ear, yet in a case, particularly like the second, it can easily be seen that where the middle ear seems to take so small a part in the process, the route of infection might possibly have been considered to be of another source. Needless to say, primary infection of the mastoid must be exceedingly rare. Just why in some of these cases the process seems to pass through the middle ear and produce so few symptoms there is still a matter of speculation. It has been attributed to the anatomic structure of the middle ear or of the mastoid bone. Among conditions of the middle ear are mentioned thickened tympanic membrane, while as causative factors in the mastoid, the sclerotic type of bone, and bones with small aditus and far forward sinus are considered.

No doubt in many of these cases the inflammation of the tympanum may be so slight and transient as to escape notice, and passes on until it reaches the mastoid antrum, where inflammatory swelling in the aditus shuts off the tympanic cavity from the antrum, with proliferation of the infective material and destruction of tissue with consequent abscess of the mastoid.

The reasons for presenting these cases are the necessity of remembering the tendency of cases of this kind to run on until intracranial complications arise.

To illustrate the value of the X-ray, as no doubt in both these cases diagnosis could have been established much earlier by this method, and the necessity of bearing in mind these cases in which typical symptoms are absent.

DISCUSSION.

Dr. McCullagh said that he had had three cases within about a week corresponding very closely to those reported by Dr. Coleman. One case gave a history of having had an earache for an hour or so which cleared up and remained so for the six weeks before he came to the office. When first seen the picture presented by the drum was one of a resolving catarrhal condition. The patient was watched for three or four weeks, without any change. He could hear a whisper at fourteen feet practically during all this time. Then one morning he telephoned, saying that he had had pain all night and that his ear was "sticking out from his head."

On examination a subperiosteal abscess was present, and on operation a large perisinus abscess was found, very much like that reported by Dr. Coleman.

A few days later another patient (a trained nurse) appeared with a subperiosteal abscess and practically the same history, except that the pain had been more marked. It cleared up under hot irrigations. The patient had had no symptoms for six weeks, when suddenly there was a rupture and perforation through the cortex. In this instance there was no erosion nor destruction of the internal table.

Dr. McCullagh said that at the same time he had another case under his care. This patient was a young man of about twenty years, who complained of a clicking noise in his ear, with a feeling of stuffiness. His drum was red, but there was no bulging nor sagging of the posterior superior wall. When he first saw this case the ear showed practically the same appearance of a resolving acute catarrhal process. The patient gave absolutely no history of any acute attack. He was watched for a month or six weeks, and then the other cases having come in in quick succession, there was some uncertainty as to what was going on in the mastoid. An X-ray picture was made, showing that the mastoid was filled with either pus or granulations. The condition was explained to the patient, and he agreed to have an operation as soon as he could arrange his business affairs, but he disappeared and had not been heard from since.

Dr. E. B. Dench said that everyone must recognize a type of latent mastoiditis in which an infection of some remote area of the mastoid occurs at the same time as the infection of the middle ear. Naturally, an acute otitis media is always an acute mastoiditis, but if—owing either to the topography of the mastoid in the individual case, or possibly to obstruction to drainage due to the inflammatory process—certain remote cells of the mastoid region are blocked off, the middle ear inflammation may clear entirely, or almost entirely, and yet a deep seated focus remain in the mastoid. He had never seen a case in which the middle ear had cleared absolutely, although in most of these cases—socalled latent in character—the remaining middle ear involvement was so slight that one hesitated in opening the mastoid. In every one of these cases operated upon, rather extensive tissue destruction within the mastoid was found. The speaker emphasized the fact that an infection confined to the middle ear alone and those mastoid cells immediately adjacent to it, is limited to a rather small space. Such an inflammatory process, if so limited, will clear up in a comparatively short time if free tympanic drainage is instituted by free incision of the drum membrane. If within ten days or two weeks from the beginning of the attack the inflammatory process within the middle ear has not practically cleared up, it is then safer to enter the mastoid process. The speaker has followed this rule for the last two years, and has never opened a single mastoid without finding the destruction more extensive than he had anticipated. With a discharging ear, or a middle ear that has not returned practically to normal at the end of two weeks, the surgeon is safer inside the mastoid than outside it.

While radiography is of considerable value, it is impossible to tell from many X-ray pictures whether this cloudiness found in the mastoid is due to softening or to a beginning sclerosis. If after a number of months the cloudiness remains and the septa are intact, we know that the condition is one of sclerosis. If the septa are broken down, then we know that the radiographic changes are due to the presence of pus.

Dr. Friesner said that what Dr. Dench had stated was very true regarding those cases where there is middle ear discharge. However, there are cases which cannot truly be called latent mastoiditis so far as its being possible to suspect them by discharge. He then cited the case of a child, seven years of age, who had been brought into the Manhattan Eye, Ear and Throat Hospital with a fulminating acute meningitis. The child had never had any history of middle ear trouble until three days before Dr. Jones saw him, and all that he complained of at that time was a slight stuffiness in the ear. The hearing was good, the temperature was normal, and the appearance of the drum was normal. When seen with this fulminating meningitis the light reflex and landmarks of the drum were normal. When the child developed a temperature, Dr. Jones did a myringotomy through what he believed at that time to be a normal drum There followed no discharge from the middle ear.

After admission to the hospital a lumbar puncture was performed. Pus and streptococci were found, and within a few hours the child died. A postmortem was done, which confirmed the antemortem diagnosis of diffuse purulent meningitis. Dr. Friesner said he chiseled through the antrum and found the tegmen soft, and the chisel dropped into a large cavity filled with mucopus. Then he shaved off the tegmen of the middle ear, and the ossicles were as clean as he had ever seen them postmortem.

Dr. Friesner said that he believes primary mastoiditis to be exceedingly rare, but this was an instance in which without any symptoms and little history of middle ear disturbance, or without anything leading one to suspect any complication, the child had an extensive destruction in the mastoid which led to the intracranial condition he had described.

He had also seen other cases. One of the surgeons at the Manhattan Eye, Ear and Throat Hospital had a trite saying: "Beware of the ear that is clearing up." Not long ago they had a case which looked like an acute catarrhal otitis, and was treated as such. Someone had done an ethmoid operation on the patient. While one of the men was examining the patient, he stopped breathing; he was operated upon, a subtemporal decompression first being done, and then the posterior fossa was examined. He had a cerebellar abscess which was opened. After the cerebellar abscess had been drained the mastoid was operated upon, streptococcus mucosus was found. The man's drum was faintly pink, no bulging; the landmarks were all present.

It is not the cases that have middle ear symptoms, etc., where one is so apt to go wrong; but where the middle ear symptoms are slight and where there is pain or headache and tenderness —that is where one must beware of ears that are clearing up.

A Fatal Case of Mastoiditis Following Pertussia and Influenza and Complicated by Meningitis and Concealed Measles.

DR. RALPH OPDYKE: Ordinarily mastoiditis follows one special infection, as influenza, measles, scarlet fever, pertussis, etc., with, of course, staphylococcic or streptococcic organisms as the exciting factor; but seldom is a patient so unfortunate as to have so many of the exanthemata, as in this instance, precede the mastoidal involvement.

C. M., aged seven years, only child, and with healthy parentage. Mother living and well. Father died accidentally several years previously. Child's history practically normal since infancy till in the winter of 1914, when she had a severe attack of whooping cough which was of a protracted form and left her in a nervous and lowered resistant condition. Hardly had she completely recovered from this when she had a severe grippe infection which greatly aggravated her bronchial irritation from the earlier pertussis and attacked her gastric and intestinal tract in such manner as to make it difficult to feed her a sufficiently nourishing diet. This naturally added not a little to aggravate her abnormal nervous high tension and make her unusually sensitive to the slightest pain or noise.

As the grippe was subsiding her ears were attacked, first the right and then the left, and the frail little girl was in constant distress. She ran a rather unusually high pulse and temperature and seemed to have more than the ordinary severe earaches. Prompt incision of the drums relieved her, as is customary, and for a time it seemed as though her troubles were over. Though the left ear made a complete recovery, the right continued discharging, and the pain in the middle ear persisted and there was some tenderness over the right mastoid. As the blood count showed a relative high infection with continued lowered resistance and her general condition failed to respond, even though the aural discharge was mixed and only a very few streptococci present, an immediate mastoid operation was done on this right side. The findings were as follows: Cells but fairly broken down, only a few drops of pus present; the lateral sinus situated well forward and, when exposed from the knee nearly to the bulb, appeared normal in every way and was not interfered with.

Patient did fairly well, but the temperature, pulse and condition, both locally and generally, did not immediately improve as is usual. Though the wound remained perfectly clean and aural discharge scant, the temperature ranged from 103.5° F. to 105° F. at nights, and from 102° F. to 104° F. mornings, with a correspondingly high pulse. Respirations stayed about thirty, and there was some cough, though the lungs were negative. Her throat kept very red and inflamed and bothered her. Cultures of same were negative for any specific germ. Measles was watched for, as she had been exposed to it a few days before the mastoid operation, but no rash nor coryza appeared, nor any other diagnostic symptoms. Another blood count was taken, showing leucocytes 15,000 and polynuclears ninety per cent. The various urinalyses were practically negative. Hiss serum was given at frequent intervals, also vaccines, but nothing reduced the pyrexia, nor improved her resistance. Some symptoms of an incipient meningitis were present, but all the various lumbar punctures were negative.

On the first of April, eight days after the mastoid operation, patient was removed home, as the advent of measles complicating was feared, the only symptoms of which was the suspicious-looking throat and the appearance of a rash after repeated hot baths. This rash disappeared and temperature again rose, and symptoms of a meningeal nature manifested themselves. Still nothing about the mastoid region gave any help, and the sinus continued to have a normal appearance, and there was no tenderness in the neck nor evidence of a thrombus being present. Several consultations by other aurists and pediatricians failed to clear up the picture to any extent, and the child gave further evidence of hidden measles with meningeal symptoms present. Her mind was perfectly clear and wonderfully alert up to within two days of her demise. Successive convulsions with cortical irritation now appeared, and a fairly typical erythema spread over her chest, face and neck. Both optic discs became injected and cloudy, and her vision failed to some extent. On the day of her death and some hours previous thereto she turned almost black with a typical advanced form of measles, and her eyes and nose gave every evidence of an advanced and tremendously severe form of infection.

During the last few hours she was in coma and her meningeal symptoms subsided entirely. No autopsy was permitted, and it is a moot question how much it would have helped to clear up the important factor as to whether the aural condition, existing for some time, had any active bearing in the later meningeal condition, or whether the latter was entirely due to the exceedingly severe measles infection which did not declare itself, but on the contrary wellnigh burned up the little body, already weakened and with no resistance from the successive infections from within and without. Certainly, during the last thirty-six hours no one could possibly have mistaken the picture of a most virulent measles, but earlier the meningeal element was not in all respects plain—that is, insofar as resulting from a local and definite cause.

As an otologist I cannot believe resecting the jugular or excising the lateral sinus would have been of any avail. It is possible, of course, that the various cerebral veins were involved from the measles poison, but certainly none in such manner nor in a location whereby operative measures would have been of any avail. Also, that everything possible was done to hasten the measles' appearance and thus relieve the absorbing poison. The appearance of the mastoid when exposed, the child's continued cachexia, the later high blood count, and protracted varied illnesses, proved beyond peradventure the patient's tremendously lowered resistance, and account most conclusively how it was almost impossible for a favorable outcome to have ensued.

Bilateral Labyrinthitis With Brain Abscess Presenting Some Unusual Features.

DR. S. MCCULLAGH: Grace K., aged twenty-seven years, gave the following history: Fourteen years ago she had had a mastoid operation performed on one side, and one year later on the other side, for a chronic suppuration following scarlet fever. Has had a right facial paralysis since that time. There is still a discharge of foul pus from both ears, more profuse on the right side. Has been absolutely deaf since the operation. For some time past has been suffering from constant headache which varies in intensity. At irregular intervals, sometimes as often as twice a month, the headache will be very severe, this severe pain lasting from a day or two to a week.

Examination.—On both ears a radical operation has been performed. On the right side there is a postauricular fistula and the tympanic cavity is full of foul-smelling pus and granulation tissue. In the left ear there is also a small amount of pus and granulation tissue. There is complete loss of function of both labyrinths. The station is very poor, even when the eyes are open, and the patient tends to fall to the right. A diagnosis of abscess in the region of the internal auditory meatus with chronic suppurative labyrinthitis was made and operation advised.

May 28, 1915, operation, assisted by Dr. A. Braun. Upon opening up the old wound and cleaning out the cavity, the external semicircular canal was found to have been eroded away. A Neuman operation was performed. The bone in the solid angle was softened and the whole labyrinth was full of granulation tissue. When the dura was first exposed there was no pulsation, but after the escape of some cerebrospinal fluid through the opening at the internal auditory meatus, pulsation was established. No abscess was found at this site. An abscess cavity, containing no pus, with firm walls, opening on the floor of the middle fossa just anterior to the knee of the sinus, where a mass of cholesteatoma had been lodged, was discovered. The opening was about a quarter of an inch in diameter and the cavity was about one inch in the vertical by one-half inch in the horizontal axis, as determined by careful probing. The opening was slightly enlarged and a drain inserted.

There was nothing of particular interest in the convalescence except the remarkably profuse discharge of cerebrospinal fluid, the abundance of which persisted for almost four weeks and did not cease entirely until about five weeks or more had elapsed. On July 4th she was discharged from the hospital and returned to her home, which is out of the city.

I saw her again on August 20th, when she gave a history of having had, for the past two weeks, "fainting spells," severe general headache, vomiting, petit mal, loss of memory, chills and a constant fever ranging from 99 2/5° to 101 2/5°. She was seen in consultation by Dr. J. A. Booth, who was of the opinion that she had a cerebellar abscess. Personally I thought that the sinus draining the old abscess cavity had closed before complete healing had occurred, or that there was a secondary abscess in that same region. The patient at first refused operation, but the pain became so severe that she consented to an exploratory operation.

On August 28th I reopened the old wound and removed a portion of the squama. Great annoyance was caused by a profuse arterial hemorrhage from between the skull and dura that necessitated such firm packing for its control as to distort the relations. An incision was made in the dura over the lateral aspect of the temporosphenoidal lobe at the site of the former abscess, and with a long, narrow bladed knife seven exploratory punctures were made in various directions with negative results. The cerebellar dura internal to the sinus was then incised and five punctures made with a like result.

The symptoms were relieved by the operation and convalescence went along uninterruptedly until September 8th. The patient had been out of bed on September 7th. Early that morning the patient complained of violent headache. The pain rapidly increased in intensity and was accompanied by screaming paroxysms. She was very restless and vomited frequently. By noon she was unconscious and opisthotonos was well marked. The pulse was poor and respiration was of the Cheyne-Stokes type. The temperature was about 99°. Dr. Booth and Dr. Braun saw her in consultation with me, and we agreed that it was a case of fulminating meningitis. No lumbar puncture was made, as I felt it was unnecessary and uncalled for. A blood count showed leucocytes, 15,000; polynuclears, seventy-two per cent; large mononuclears, three per cent; small mononuclears, twenty-five per cent.

September 8th. Condition unchanged. Maximum temperature, 101°. Respiration, 14 to 30; pulse, 90 to 118. Blood count: Leucocytes, 28,260; polynuclears, ninety-two per cent; large mononuclears, six per cent; small mononuclears, two per cent.

September 9th. Patient conscious. Pain in head not severe. Vomiting persists, perhaps due to the morphin she had been receiving in large doses.

The nurse who had attended her after her first operation happened in and gave a history of a similar attack at that time, when the patient had been unconscious for four days with a well marked opisthotonos.

During further convalescence there were two similar but less marked attacks, accompanied by severe pain with screaming, vomiting and slight rise of temperature. The first attack lasted about five days, the second about two.

The patient was discharged from the hospital on October 4th.

I saw her last on February 4, 1916. The ear was completely healed. She still complains of a constant dull pain in the right temporal region, localized about one and one-half inches above the ear and over the right eye, in the region of the frontal sinus. She has attacks of what she describes as severe pain which come on at irregular intervals, have an indefinite duration—"usually all day"—and subside gradually. Otherwise she is well.

The only hypothesis that would seem to give a rational explanation of this history is that drainage from some undiscovered intradural focus of infection was temporarily shut off, precipitating the crises mentioned.

DISCUSSION.

DR. BLACKWELL asked if the abscess cavity had a wall.

DR. MCCULLAGH replied in the affirmative. It was evidently an old abscess with good rigid walls, which they were careful not to disturb.

Paper: Brain Abscess of Internal Capsule Following Injury to the Lateral Sinus During Mastoidectomy—Operation—Recovery.

BY WELLS P. EAGLETON, M. D.

DISCUSSION.

DR. DENCH expressed the opinion that so much had been said in the paper that there was little left to discuss. However, owing to the fact that Dr. Eagleton had been obliged to abridge his presentation of the case, one or two points were not quite clear to the speaker.

First.—He would like to know what the microorganism was which caused the trouble.

Second.—As he understood the history, the labyrinth was normal at the beginning of the attack, and thirty-six hours later was dead. (Dr. Eagleton stated that this was the fact.)

Dr. Dench was of the opinion that the serous labyrinthitis in this case was probably of mechanical origin, and was probably due to the sudden occlusion of the lateral sinus by a clot. This in turn might give a back pressure in the labyrinth and be followed by an effusion of serum. Dr. Dench thought that as at the first operation the abscess was opened but not drained, the subsequent relief was very largely due to the decompression effect of the first operation. He could not quite understand how the patient escaped a purulent meningitis if the abscess was opened but not drained until six weeks later. At the second operation the drainage of the abscess through the center of the osteoplastic flap seemed to the speaker to be a procedure of distinct advantage. The speaker then detailed briefly a history of a metastatic abscess occurring in the island of Reil, in a case upon which he had operated and where the sinus had been opened at the time of the original operation. In this case the symptoms of involvement of the island of Reil came on about five weeks after the primary operation. The sudden occurrence of aphasia in the case, together with a high polymorphonuclear count, seemed to point to an abscess. Dr. Dench was urged, however, by the attending physician and by the consulting neurologist not to operate upon the patient, as these gentlemen looked upon the case as one of thrombosis. The operation was afterwards performed, too late, and although the abscess was drained the patient died. At the time of the operation the abscess proved to be an old one, as the walls were exceedingly firm. This patient gave a history of having had an attack of acute otitis ten years before. The speaker believed that the original purulent focus was deposited in the neighborhood of the island of Reil at the time of the first acute otitis, and that the second infection, ten years later, followed by the mastoid operation and the opening of the sinus, simply led to a lighting-up of the activity of this latent purulent focus in the speech area. The speaker's reason for believing this was the fact that well marked symptoms referable to an abnormal condition in the speech area developed every time the patient took an anesthetic; that is, the original attack ten years before the death of the patient occurred in Italy, and whenever the patient was given a general anesthetic she would immediately begin to sing songs in the Italian language, a language with which she was only slightly familiar and which she had practically not used since her visit to Italy, ten years before.

Dr. Scruton said, in speaking of Dr. Eagleton's paper, Dr. Dench had said that the operation was practically a decompression, which Dr. Eagleton denied. He agreed with Dr. Eagleton in his position, for the reason that edema occurs whenever the brain is exposed, and if, as in this instance, the bone flap is replaced and the scalp sutured, it is not conceivable that much if any decompression is accomplished. The relief of symptoms in this case was entirely due to the evacuation of pus from an abscess cavity, and not to the lifting of the bone flap. The subsequent history bears this out as fact.

NEW YORK OTOLOGICAL SOCIETY.

Meeting of March, 1916.

Hemorrhagic Measles.

Dr. Philip D. Kerrison described a severe and unusual case of hemorrhagic measles which he had seen the same day in the Willard Parker Hospital. The patient, a girl of twelve years, came from a tenement apartment in which three other children of the same family had measles of the usual type. The patient under discussion was removed to the hospital on account of the unusual features and apparent virulence of the disease.

She had been ill three days, and there was a history of hemorrhage from the bowels (with the stools) and rather profuse hemorrhages from the nose, to control which both nostrils had been packed with sterile gauze. When admitted to the hospital she was also bleeding from the right ear. The ears had not previously discharged.

When seen by Dr. Kerrison the temperature was 103° and the patient looked profoundly ill. The skin from head to feet was covered with closely approximated reddish mahogany spots. In the mouth hemorrhagic spots were seen only on the roof. Both nostrils were packed with gauze. The right ear showed coagulated blood upon the drum membrane, which he did not remove. The left drum membrane presented an appearance which he had not seen in any other patient. It was intact, but showed the color changes due to blood filling the tympanic cavity. That is to say, the membrane, though reddened by contact with blood filling the tympanic cavity, did not appear to be inflamed, and the patient did not complain of ear pain.

Dr. Kerrison said that he had not incised either membrane, and had left instructions that the ears be left alone unless symptoms characteristic of tympanic inflammation developed. It was in his experience an unusual case, the outcome of which he would be able to report later.

DISCUSSION.

Dr. Harris said he saw bleeding from the ear a little over a year previously, occurring in an adult. The man had a history of repeated hemorrhages, before he was brought to his office, from the gums and from the nose. He was sent to the hospital and twenty-four hours after he had entered the patient had a very severe hemorrhage, and when Dr. Harris got to the hospital he found him exsanguinated and with hemorrhages in both ears. Examination of the drums showed, as far as he could make out, not trying to meddle too much with the condition, that both middle ears were filled with blood and the hearing very much reduced. The patient later had characteristic subcutaneous hemorrhages all over the body. The case was plainly one of purpura hemorrhagica. He tried to hunt for the source of the infection, and much to his surprise the bacteriologist succeeded in getting a pure culture of the Klebs-Loeffler bacillus from the tonsil. The conclusion Dr. Harris arrived at was that this case was one where the diphtheria was latent and yet where there was a sufficiently active organism in the tonsil to give rise to the blood changes and so to the hemorrhages. The process in the ear was allowed to take care of itself. The patient was given fifty to one hundred cubic centimeters of human blood serum, which promptly stopped the bleeding, but it must have taken two or three weeks for the blood in the ear to become entirely absorbed. Before he left the hospital the hearing was fully restored.

Dr. Dench asked whether this case did not resemble cases of hemorrhagic otitis complicating grippe. In grippe it was not uncommon to find hemorrhagic blebs in the canal, upon the surface of the drum membrane, and even hemorrhage into the middle ear. In some of the cases which went on to mastoid involvement, the mastoiditis was of the hemorrhagic type. Dr. Dench had always felt that it was the severity of the constitutional infection producing a toxin which was responsible for the hemorrhagic form of otitis in grippe. A similar condition might be found in diphtheria, scarlet fever, or any other severe constitutional disease.

Dr. Kerrison said that his case showed an absolutely different picture. It was hard to describe it, but the case had no resemblance to the grippe ear. The blood was examined and there was not much change to be found.

DR. HARRIS said that in his case there had been decided blood changes, with delayed coagulation and all the other evidences which Dr. Dench had pointed out.

DR. DENCH asked Dr. Kerrison if there had been any delayed coagulation in his case; if so, he would like to know what the time of coagulation had been.

DR. KERRISON said that the delay in coagulation had been very slight.

Tubercular Mastoiditis.

DR. ROBERT LEWIS said he wished to relate the history of a case which was one of three similar cases, all of whom were born in the same institution within a few weeks of each other, in October. The three cases had been seen by different men, all had been operated upon and all had had a radical operation performed, which was in each case unfortunately followed by a facial paralysis. His case he saw in the latter part of December; the child, three months of age, had had a running ear for some few weeks before she was first seen by him. It was impossible because of the stenosis of the external auditory canal to see the drum membrane. There was a discharge coming from the ear of a thin bloody serum. He suggested operation at the time, but the mother objected seriously. He saw the patient at intervals of about two weeks for nearly six weeks or two months, and every suggestion of operation to the mother was met by refusal. Dr. Lewis advised her to obtain another opinion. She consulted Dr. Whiting some time later about her baby, and he advised immediate operation. A von Pirquet test, made by the family physician, gave a positive reaction. Dr. Lewis was then asked to operate upon the case.

He found the mastoid process filled with a mass of cheesy material, granulation tissue and necrotic bone; the posterior canal wall came away as a sequestrum. Very little curetting was done around the facial canal, but after the operation the child had a facial paralysis. It could close the eye, but in crying there was a distinct paralysis to be seen. The other two cases were also cases of tubercular mastoiditis. There was a cortical perforation and the periosteum covering the process was very thick, almost like cartilage. There was no evidence of a subperiosteal abscess.

Dr. Lewis said that this child had been partially fed by the bottle, and that the institution was very careful about the source of its milk.

DISCUSSION.

Dr. Dench said he would like to ask whether or not the facial paralysis had been present in the child before the operation was performed.

Dr. Lewis said he lifted the posterior canal wall right out, and this presumably exposed the nerve. From the fact that there was only slight paralysis of the upper portion of the face, he hoped it would eventually clear up. The child was three months old when he first saw it, and had been operated on only eight days previously. All of the middle ear was filled with necrotic bone and granulation tissue.

Dr. Berens said that sequestrums were very dangerous things in these cases, as one never knew how much of the nerve was exposed. He asked whether the evidence was that of a general tubercular infection or whether it was only local.

Mastoiditis—Vaccines.

Dr. William H. Haskin said he recently had a very interesting experience in a child, where he had operated for mastoiditis in both ears, one year previously, which he believed had been reported before the society as a case of infection having come from an abscess in the jaw. The child became very well, both mastoids healed up beautifully, and the drum membranes were intact. In the latter part of December the child had an attack of grippe and an acute otitis media in the right ear. The drum membrane was incised and the patient got well. In the latter part of January the patient had an acute otitis in the left ear, but because of the mother having become ill the case was neglected for a week or ten days, when she showed a big posterior swelling. The drum membrane was bulging, but there was no breaking at all of the drum. Dr. Haskin made an incision through the membrane and behind the ear, but found the old mastoid wound filled with healthy looking bone, and he thought that pus had burrowed up along the superior wall of the canal. He did not do any further operation on the bone and watched her for a little while. A great amount of edema remained over the mastoid and cultures were taken, a staphylococcus infection being found. The child received some injections of autogenous vaccine, and after the third injection the whole condition quieted down, the ear returning to its normal condition. Later the mother again brought the patient to Dr. Haskin, and this time the right ear was affected. The child was out playing in the snow and had gotten chilled, an acute infection having followed after two or three days. There was some swelling behind the auricle. The drum membrane was opened and the child immediately started on vaccines again. Much to Dr. Haskin's delight, after the third or fourth injection that ear completely recovered. He had seen the child at intervals of a week ever since, and there has been no further infection of any kind. The child had had ethmoiditis at the same time, which was treated with argyrol and suction. Dr. Haskin said he had been strongly tempted to go in behind that ear to operate, but did not think anything would have been found if it had been uncovered.

DR. BERENS said he would like to ask Dr. Haskin if there was no pus in the superficial tissue at all in this case, and if it was a socalled "white swelling."

DR. HASKIN said there was no pus, but a great inflammatory thickening. He said the swelling above the mastoid wound over the upper portion of the canal was a red and not a white swelling. He had fully expected to find pus, but did not.

Chronic and Recurrent Mastoiditis in Infants.

DR. E. B. DENCH said that within the last two years he had operated upon quite a large number of infants at St. Luke's Hospital suffering from recurrent or chronic mastoiditis. In some of these cases there was a history of intermittent discharge, while in others the persistence of the discharge was the cause for operative interference. In these cases the outcome had been eminently satisfactory, in marked contrast to the prognosis in mastoiditis in young infants given by Dr. L. Emmett Holt. In many of these young infants the operations were done not primarily for the relief of the condition within the mastoid, but simply because the children were in a marasmatic condition and no other cause could be found for the marasmus excepting the aural suppuration. In all of these cases, when the mastoid was opened, it was found to be the seat of a suppurative process, filled with granulation tissue and softened bone. Immediately after the operation these children uniformly began to gain in weight. The same flattering results have followed the radical operation in young children where a profuse purulent discharge had persisted, and where the discharge itself, as well as the poor general condition of the child, was an indication for operation. In some of these cases, primary grafting had been followed by excellent results. In others, where the local condition was too foul for primary grafting, secondary grafting had been followed with benefit.

The speaker called attention to the fact that in many cases, especially in children, the middle ear lesion might clear up completely and a focus of infection remain in the mastoid antrum. This condition explained, undoubtedly, the many attacks of recurrent acute otitis with high temperature, so frequently seen in children. He also explained the cases of so-called fulminative mastoiditis sometimes observed—that is, cases with a history of acute otitis, with severe symptoms demanding mastoid operation within twenty-four hours after the middle ear became involved, and where upon opening the mastoid extensive destruction of the mastoid structures was found. The extensive destruction of the mastoid was undoubtedly not due to the immediate acute process, but was the result of several recurrent attacks of otitis, each attack causing a little more destruction within the mastoid until finally operative interference became necessary.

Dr. Berens said he would ask if any anatomic peculiarities had been found in any of these cases, and what the infecting organism shown had been.

Dr. Dench said that these cases usually showed a streptococcus to be the infecting organism.

Dr. Quinlan asked was not the infection through the pharynx; as many mastoid operations had been performed and the condition of the rhinopharynx had been overlooked and the culture field remained, it was likely that the infection could have easily been transmitted through the tube (if such was not obliterated at the time of the mastoid operation). Dr. Quinlan thought that the cleaning up of the pharynx had as much to do with success of the work as the major operation on the mastoid; even after the tonsils and adenoids had been removed, one must bear in mind that bad teeth had much to do with these infective conditions.

Dr. Dench said he wanted simply to call attention to these cases, as he had not seen anything written about them.

Dr. Haskin said the discussion bore out the same line of thought which was much in vogue about the focal infection of teeth. These patients with a focal infection invariably had a lowered vitality and marked anemia.

Dr. Berens said that at the Manhattan Eye, Ear and Throat Hospital the men operated on these recurrent cases in children without fear, and had not been scared particularly by bad prognosis. The results were excellent.

Osteomyelitis.

Dr. Wells P. Eagleton said that some time over a year ago he presented a man, reported as a case of osteomyelitis, who was also presented before the Eastern Section of the American Medical Society. At that time he was asked by Dr. Dench if a Wassermann test had been made, and Dr. Dench told him that he thought the case was one with a syphilitic process from his experience of such cases. Dr. Eagleton told him he would have a Wassermann made. The man is now perfectly well, there was no return of the ear trouble, the incision had entirely healed and there was very little scarring. About one month previously Dr. Eagleton wanted to publish the case, but got hold of the patient's physician before he put the matter in print and asked this doctor to tell him how the patient was. The man was the father of three healthy children. The Wassermann had been three plus. Dr. Eagleton said he would not publish the case as one of osteomyelitis.

Cerebellar Lesions—Specimens.

Dr. Wells P. Eagleton said that four months previously he had presented a specimen before the society, which he was now again showing as a sample of some of the middle ear symptoms that occurred in the pontal cerebellar angle tumors. During the last month there had come under his observation another such case, in which the otologic symptoms had also been thoroughly studied over a period of several months with the idea of a diagnosis. Dr. Eagelton said the one which the members had already seen emphasized the points of importance. The one cerebellar angular tumor was a dural endothelioma. The man, prior to operation, had a marked nystagmus with the recognized tuning fork reactions. This case had been under observation altogether for a period of two years. There was very much doubt about the diagnosis of tumor in the second case, that of a child. Two very competent neurologists thought it was a case of hydrocephalus. At the autopsy a tremendous tumor was found. At no time did the child present any nystagmus, illustrating the point that they only had nystagmus in extracerebellar lesions and not in true intracerebellar lesions. Dr. Eagleton said that in a paper read before the American Triological Society three years previously, he called attention to the fact that in the vestibular reactions the opposite ear would be affected in cases of cerebellar growths. This child had been subjected to caloric reactions twelve times during a year and a half. At times there would be in the right ear an absolute absence of the reaction to the caloric. There would be no dizziness, no nystagmus, and the child would walk off without any symptoms at all. At other times, however, the reaction in one ear or the other would be very marked, and this would depend upon the condition of the child's head, as if the degree of the hydrocephalus had something to do with it.

During some days it would be suffering intensely. When the child was in good condition a reaction to the caloric could be produced from either side. Dr. Eagleton said this fact was interesting if it would tally up with some of the other otologic symptoms of the intracranial groups.

DISCUSSION.

Dr. Dench said he thought the loss of caloric reaction in these cases was due to increased pressure of the cerebrospinal fluid. The perilymph and endolymph communicate directly with the subdural space and with the ventricles of the brain. The speaker thought that in cases of hydrocephalus the ventricular fluid would be dammed back, and consequently, free circulation of the labyrinthine fluid would be prevented, thus accounting for the absence of the caloric reaction. He had seen two cases of cyst of the frontal lobe in which there was an absence of caloric reaction upon the same side. This absence of caloric reaction might be explained either by noncirculation of the cerebrospinal fluid or by an edema of the vestibular nerve, due to pressure; in other words, we might have an edema of the auditory nerve or labyrinth, exactly the same as we have swelling of the optic nerve in cases of increased intracranial pressure due to tumor.

Regarding the remarks that tumors within the substance of the cerebellum did not cause nystagmus, he felt certain that he had seen cases of intracerebellar tumor in which nystagmus had been present. A recent author had stated that in cases of a tumor within the substance of the cerebellum, this symptom was not produced until the tumor grew to a sufficient size to become extracerebellar as well. Dr. Dench had no experience to confirm this statement.

DR. HASKIN said he would like to ask Dr. Eagleton what the nature of the tumor in his last case had been.

DR. EAGLETON said that the tumor was a glioma, although it had not been microscopically examined.

He said he thought otologists could be of assistance to the neurologic surgeons, as the caloric reaction, if applied properly, would help them add their mite of evidence toward the diagnosis of increased intracranial pressure, even without localized cerebellar symptoms.

DR. DENCH said that in certain cases decompression effects were certainly obtained simply by removing a portion of the calvarium without opening the dura. English observers denied that any decompression effect was possible without incision of the dura, and had performed certain experiments upon the dural sac (namely, attempting to dilate this with water) to demonstrate its absolute inelasticity. Notwithstanding these experiments, however, Dr. Dench was certain that in some of the cases where a large portion of the skull had been taken away and the dura had not been incised, a certain amount of decompression effect was obtained. He was very glad to agree with Dr. Eagleton in this regard.

Diphtheria—Hemorrhages—Mastoiditis.

DR. FREDERICK WHITING said he had recently had a case in a child, with an involvement of both ears. There was suppuration from both ears with a very large mass of glands under each ear and in the neck. The patient was running a very high temperature and was just such a case, Dr. Whiting said, as he was sure most of the men present had at least once during the winter. There was a question as to whether the case with which he dealt was a simple middle ear condition or mastoiditis and sinus thrombosis. The temperature was of a fluctuating character and varied from 99 to 105 degrees, and the patient's appearance gave them great concern. The cultivation of the blood was negative, so Dr. Whiting concluded there was not a sinus phlebitis. After a reasonable length of time the physical appearances were such as to indicate the presence of mastoiditis, and a double mastoid operation was performed. The child did not seem to do well. The mass of glands in the neck appeared to diminish in no respect, and there appeared on the child's face and on the extremities quite a number of large ecchymotic spots. These spread and in the course of thirty-six hours assumed proportions which were anywhere from the size of a silver dollar or larger down to very small hemorrhages. The question arose as to whether or not the child might have scurvy, and in an examination of the mouth and gums Dr. Whiting discovered a small patch on one tonsil. He said that the throat had been examined day after day and found negative; but that morning in looking for hemorrhages on the gums, which he did not find, with the exception of one or two small ones, he saw the patch on the tonsil, which was gray and very suggestive. A gelatine, health-board culture tube trial was made, and by night there was a large gray patch on the other tonsil. When the report came back it was said to be the Klebs-Loeffler bacillus.

The hemorrhages in the meantime had extended pretty much over the body. No attempt was made to count them, but they appeared in great numbers, over the body, backs of the hands, arms, legs, face, palms, etc. The mastoids, as far as physical appearance went, looked very well; they were not granulating very freely, but there was no evidence to lead one to believe that such granulations as were forming were losing vitality at all. It was decided to do a transfusion in the case.

Dr. Lindeman of Bellevue did the transfusion, and a college classmate of the father of the child volunteered to supply the blood. This blood was replaced by a double amount taken from the donor.

The next morning it was with difficulty that one could recognize the presence or situation of any of the hemorrhages that had been present the day before. In forty-eight hours there was not one to be seen, and in seventy-two hours one could not tell that the child had ever had any. The child's general condition improved in proportion to the skin condition; it went through a moderately quick recovery from the diphtheria, for which antitoxins had been administered, and although the adenitis persisted for three weeks or more, the patient finally made a perfect recovery and was discharged cured.

One of the features of the case, Dr. Whiting said, which was of rather remarkable coincidental interest, was the fact that the year previous this child's cousin, a boy of the same age, developed a double mastoid and diphtheria, and he had operated on him for double mastoiditis when the child had the tube in his throat. It was merely a coincidence, but seemed a most astonishing thing that the sons of two brothers, at intervals of exactly one year, should both have been subjected to a double mastoid and diphtheria, although the second one did not have a purpura.

DISCUSSION.

Dr. Dench said he would like to ask Dr. Whiting whether he thought the glandular enlargement entirely due to the diphtheritic infection.

Dr. Whiting said that the glandular enlargement had been present fifteen days before the diphtheritic infection. The throat had been examined every single day because of the temperature, and never had presented any appearance which would suggest it microscopically. There was nothing in the throat, and of course as soon as he had seen the spot on the tonsil, a cultivation was made.

Dr. Dench said he was inclined to think a good many of these cases of enlarged glands coming on after middle ear infections were cases of sinus thrombosis where one failed to have bacteremia. Large glands over the bifurcation of the jugular vein, just at a point where the facial vein goes off from the internal jugular vein, it seemed to him, if there were a rise in temperature, might be a sign of a parietal clot. In a case he had seen, in which the child had not been operated on, although it actually got well, Dr. Dench thought there was a parietal clot in the sinus. He said a certain number of these cases of sinus thrombosis did get well without operation, but there was no reason to suppose that a small parietal clot could not have existed in the sinus. Gland enlargement was really a life-saving measure, because it filtered out the germs from the blood.

Dr. Whiting said during the winter he had had two cases of sinus thrombosis. They were both cases which came into the Mount Sinai Hospital at night, and presented the ordinary appearance of a subperiosteal abscess. Both of them were in children, one four years of age and one about three. Dr. Whiting said he was called and the cases were operated on before there was time to find out much of their previous history. They came in with a high temperature, a blood cultivation being made before the operation. In both of these cases, because of the very high temperature, Dr. Whiting opened the inner table and inspected the sinus, which appeared perfectly normal.

The next day, twenty-four hours later, both cultures were reported positive. The temperature in the meantime had dropped. As the children seemed to be in excellent condition, Dr. Whiting thought he would give them a chance and watch them. The next day the temperature did not rise, did not go above 102 degrees, so he thought he would wait another day, and so tided them along. Both cases made an uneventful recovery. Neither of these cases, bearing on what Dr. Dench suggested, had any large glands; certainly nothing which was enough to attract attention.

In view of these results, it would appear that the last word has not yet been said concerning the value of positive blood cultures in children.

Dr. Dench said he would like to ask if the tonsils had been previously removed in that case.

Dr. Haskin said that he had two patients at the Manhattan Eye, Ear and Throat Hospital, bearing out the same experience which Dr. Whiting had had, and which showed positive bacteremia, following mastoid operation, streptococcus hemolyticus, and he had asked Dr. Dwyer to operate on one of the patients. The patient developed a very severe erysipelas following operation. During the operation the sinus was not opened and the patient ran a temperature of about 105 degrees for several days. They had found two positive blood cultures, although positive blood cultures were not usually found in the erysipelas condition. Dr. Haskin advised Dr. Dwyer to let the patient alone and the condition subsided, the erysipelas got well, and the patient was now alive without any trouble whatever.

Dr. Culbert had another patient which he operated on with the same history of a temperature of 105 degrees following operation. A blood culture was made, which was simply swarming with streptococcus hemolyticus. On the following day it subsided. A second culture was made four days later, but no bacteremia were found. He said he thought Dr. Berens had given orders that all cases of acute mastoiditis should have blood cultures taken, regardless of any indications for sinus thrombosis or not. That rule had been carried on for some time at Dr. Rae's clinic. He said he did not know the exact number of bacteremias, but knew that there were a number where the sinus had not been operated upon, but where the cases had recovered.

Dr. Eagleton said that Dr. Libman, who had contributed so largely to this subject, had told him that he had had one case where a blood culture was made, which after forty-eight hours was announced positive. The child recovered without further operative procedure. Dr. Eagleton said that a chill had occurred a number of times in such cases, and as far as he could make out, it uniformly occurred in children.

Dr. Duel said he would call attention to the fact that several years ago Dr. Jonathan Wright and he, at the Manhattan Eye, Ear and Throat Hospital, had made examinations for bacteremia, which they found present in a number of cases of acute otitis, chronic otitis and operated cases of mastoiditis not demanding further operation. He believed that bacteremia being present in many cases did not necessarily mean sinus thrombosis, nor did it demand jugular resection and sinus obliteration, in order to save the life of the patient. Often, on the other hand, after careful search, the bacteremia was never found, which it was thought must be present in suppurative conditions, the clinical manifestations of which so clearly indicated most radical surgical interference.

Dr. Berens said the bacteremias were not present in the demonstrable clot. This stand was the one that several others took, together with Drs. Jonathan Wright, Duel and Libman, who had reported these investigations at the Otological Section of the Academy of Medicine.

Dr. Duel said he thought that Dr. Wright had the right opinion at the time of their investigation. He thought that the pouring out of the bacteria into the blood, producing bacteremia, was followed by a tendency of the blood to immediately demolish these bacteria. He believed it was not the tendency of the blood to form a culture medium for the reproduction of more and more bacteria, but that it was only from the local focus of infection that the bacteremia took place, and that one might frequently try a culture in a case where a bacteremia was actually present without demonstrating it. Only in certain very virulent types of infection would the blood not take care of the condition within a very short time. It, therefore, depended on the time a culture was made, the vein from which it was taken, and the amount of blood removed.

Dr. Whiting said that Dr. Wright's proposition must contain a good deal of truth. He had operated on three cases during the past fifteen years, all of which were thirty years of age or over, and each one of whom gave a history of having had previous ear disease in childhood. When he operated he found an osteosclerosis, the densest portion being just over the situation of the antrum, where there had undoubtedly been a perforation of the cortex many years before, and where subsequently the wound had healed. There had been no operation of any kind, and the patients had come from that class of people who, if they were not operated in the hospital, had no other recourse for operation. The bone processes had healed. Each of these patients had come for operation many years after the earlier attack of mastoiditis.

Each of these cases had an epidural abscess, and when he cut away the skull sufficiently to expose the bed of granulating dura, he found the lateral and sigmoid sinus nothing but a firm, hard, fibrous cord.

The sinus had been obliterated for many years. Dr. Whiting considered that in the attacks in early childhood there had been spontaneous healing of the mastoid abscess through perforation of the integument, and that the sinus thrombosis had undergone spontaneous cure.

Dr. Dench said that undoubtedly certain cases of sinus thrombosis recovered spontaneously, with total obliteration of the lateral sinus. He had seen at least one case of this kind in which no vestige of the sinus remained. Dr. Day, of Pittsburgh, had reported a series of these cases last year—either before the American Otological Society, or the American Laryngological, Rhinological and Otological Society.

Regarding Dr. Duel's statement, it was interesting to note that sometimes a blood culture from a vein in the arm would be negative, and yet if a culture were made from the infected lateral sinus this culture would be positive. (Dr. Dench had seen a case of this kind.) In other words, the blood possesses certain bactericidal properties, and although the blood contained microorganisms in the immediate vicinity of the local focus of infection, these organisms disappeared, owing to the bactericidal properties of the blood, in their passage through the general circulation. Dr. Dench pointed out the fact that it was not necessary to have a demonstrable clot in order to have bacteremia. In all cases of sinus thrombosis the infection begins in the outer wall of the sinus and then passes to the inner wall, a parietal clot forming at this point. A bacteremia would occur while the clot was parietal, but when the vessel became completely thrombosed the bacteremia would disappear—returning again only with the breaking down of the clot. It was then time to call in an undertaker, not a surgeon.

Dr. Duel said that, after all, we were coming back to the position of Dr. Gruening, who first called attention to these cases. He had said he operated on the clinical indications rather than on the laboratory reports. Frequently a case showed bacteremia which presented no alarming clinical manifestations and recovered without operation. Another case might be taking in a very small number of very virulent organisms and showing many urgent clinical symptoms which demanded obliteration of that portion of the vein near the infected area, whether a clot was preesnt or not. The arrest of the circulation in such a sinus was clearly indicated, to prevent general infection.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Regular Meeting, Held December 21, 1915.

DR. GEORGE W. BOOT, THE PRESIDENT, IN THE CHAIR.

Case of Atrophic Rhinitis Following Removal of Turbinates.

DR. GEORGE W. BOOT showed this patient for the purpose of getting an expression of opinion from those present regarding the class of work represented. The man was at some hospital in Chicago, where he paid a dollar and a half for a certificate, and he was to be taken care of if sick. He went to the institution with nothing very serious the matter, and they persuaded him to have an operation on his nose. His turbinates were removed, and they managed to extract fifteen dollars from him for the work. After leaving the hospital he was taken with a severe hemorrhage and brought to the County Hospital after he had lost a considerable amount of blood. At present he has a condition of operative atrophic rhinitis. He has a tinnitus in the right ear, which he did not have before operation. Dr. Boot's attention was attracted particularly to this case, because previously he had been very nearly involved as an expert witness in a malpractice suit in a similar case.

He wished the members would express their opinions as to what should be done in such a case; whether to keep still and let these men go on deforming noses in this way, or try to do something to have it stopped. In this case both lower turbinates had been very completely removed, especially the left side, followed by crusting.

DISCUSSION.

DR. JOSEPH C. BECK asked the speaker if he claimed an atrophic rhinitis in consequence of that operation.

DR. BOOT replied in the affirmative—an operative atrophic rhinitis.

DR. JOSEPH C. BECK thought an individual expression of opinion might be given. His opinion is that atrophic rhinitis does not result from the removal of the inferior turbinates. In the days when the inferior turbinates were removed for all sorts of conditions, particularly ear conditions, he had seen something like forty cases while visiting in Louisville, in Dr. Dudley Reynolds' practice, in which he (Dr. Reynolds) had removed lower turbinates on both sides. These cases were all the way from one to three years after operation, and in none of them was there any crusting in the nose, or anything that looked like atrophic rhinitis, that we are accustomed to speak of as such. They all had more or less pharyngitis sicca, with the deep injected and glazed smooth posterior wall of the pharynx. They did complain of lack of air rather than too much air, but he thought all operators had had that experience with these cases. As to removal of the lower turbinates as a causative factor of atrophic rhinitis, he would take issue with Dr. Boot.

Dr. Alfred Lewy did not see the case, but from a medicolegal standpoint, he would like to refer to a case he saw in which both lower turbinates had been removed by one of the best known general surgeons in Chicago. In this case the patient was troubled with persistent crusting for many years after the operation.

Dr. A. H. Andrews thought it would be interesting to know the condition of the nose before the operation in Dr. Boot's case. If there was atrophic rhinitis soon after the removal of the inferior turbinates, he would be inclined to think that the same process existed before they were removed. He does not believe in the indiscriminate removal of the inferior turbinates. Such practice should be severely condemned. With Dr. Beck, he questioned whether real atrophic rhinitis is ever caused by the increased lumen of the nostril. It requires other etiologic factors to produce the pathology.

Dr. J. Holinger thought that a Wassermann and a systematic washing of the cavities would be of importance in such a case before making the diagnosis of atrophic rhinitis. There are scars in the nose that seemed to him to have the typical star shape so characteristic for scars after syphilitic lesions.

Atrophic rhinitis is essentially different from a specific rhinitis, or rhinitis due to suppuration from different cavities. These diseases ought to be excluded before a diagnosis is made, since either can cause crusting.

Dr. Boot, in closing the discussion, said he did not mean to give the impression that he thought the pathologic changes of atrophic rhinitis were present in this case. It is a case, however, where there is a very marked condition in the nose, as a result of the operation, and he has some of the clinical signs of atrophic rhinitis. He did not mean to say that it was an atrophic rhinitis from a pathologic standpoint.

In his opinion, the case certainly does not resemble a case of syphilitic ozena.

Dr. Boot, in the absence of the other two cases which he had expected to demonstrate, exhibited a specimen which he thought would be of interest. It was removed postmortem from a man of about thirty-five years of age, who had been in the County Hospital off and on for a year or two. He entered first with the diagnosis of syphilis of the larynx, and was under Dr. Friedberg's care. He recovered sufficiently to leave the hospital, but returned after a year or so. At this time he was very dyspneic, and it was necessary to do an emergency tracheotomy. It was never possible to dispense with the tracheotomy tube. After wearing it for about a year he came back again, desiring to have somthing done so that he could breathe through the larynx. He could not get any air into the lungs. He could exhale and phonate, but no air would go in. On examination, the speaker could not find an opening through the larynx, and was unable to pass any sound through, so he went in in the other direction—through the old tracheotomy wound—and as he supposed cut into the larynx, but cut into this cavity (illustrating on specimen). Very shortly after this, before he had succeeded in dilating the stricture, the man began to cough out any substance he would drink through the tracheotomy tube, and he was then turned over to the general surgeons for a gastrostomy, so that he could be nourished. He did not rally from the operation of gastrostomy, but died on the third day, and at the postmortem the specimen was removed. The speaker passed a toothpick through the opening from what he supposed was the lumen of the larynx at the time of the laryngofissure, but which may or may not have been that. At the postmortem the posterior part of the larynx was found in this position, and an opening below was found where the tracheotomy tube had ulcerated through. He has been unable to find the arytenoid cartilages in the specimen, and not much of the thyroid cartilage. An interesting feature of the case was that at the postmortem he cut into the thyroid gland and found a round, whitish area, which he supposed was a gumma. A piece was cut out for sectioning, and afterwards reported to be a carcinoma.

Carcinoma of the Mandible—Exhibition of Specimen.

Dr. L. W. Dean, Iowa City, Iowa, exhibited a specimen of squamous celled carcinoma of the mandible, without any ulceration of the mucous membrane of the tongue, floor of the mouth or lips. This condition was operated on by the method devised by Dr. Prentiss for the removal of half the mandible without interfering with any of the branches of the facial or producing any deformity about the mouth. An interesting feature in connection with the case is the appearance of the patient at the present time, shown in the photographs passed around.

This patient came with a history of having had, in October, 1914, a small, warty excrescence removed from the angle of his mouth. Following this operation the wound healed quickly. The speaker was unable to get the excrescence which was removed for microscopic examination. Naturally, he assumed that it was this squamous celled carcinoma. One month after its removal the patient had a tender premolar. The excrescence was on the right side and the tender premolar was in the mandible of the right side. Almost all patients with malignant growths in the mandible, and many of those with malignant growths in the superior maxilla, give a history of having had teeth extracted for pain, and the dentist telling them that the tooth was normal. This patient had this premolar extracted a month after the operation on the excrescence, and the dentist said it was a vital tooth, so the speaker assumed that this malignant growth was developing at that time in the center of the mandible. Following the removal of the tooth the patient had no trouble until last June, when he felt a little nodule on the border of the ramus, below the premolar on the right side.

When the patient came under Dr. Dean's observation he had a growth of some size—probably the size of half a hen's egg—which was not tender on pressure. It was very hard. Wassermann examination negative. X-ray examination showed absorption of bone. There was no ulceration at this time of any of the mucous membranes of the mouth. Since seeing this case, Dr. Dean has gone over the literature on the subject very carefully, and has found the statement frequently made that whenever you have a squamous celled carcinoma of the mandible, you always have an ulceration of the mucous membrane, either of the tongue, floor of the mouth, alveolar process or lip. The fact that there was no ulceration of any of these membranes at the time would suggest that it was not carcinoma. A piece of the growth was removed, however, after permission for operation was secured, providing it was found to be malignant, and examination showed it to be a typical squamous celled carcinoma.

In the one patient from whom Dr. Dean had previously removed half the mandible there was the usual postoperative drooping of the corner of the mouth and some flow of saliva over the chin and noticeable deformity. He consulted Dr. Prentiss regarding the operation. Dr. Prentiss advised the following operation, which was performed: Incision in the median line, beginning at the lip, going down over the chin, and then lower down on the neck, extending backward and upward, and terminating at least three inches below the ear. This incision extended through the skin, superficial fascia and platysma. These structures were lifted up to the inferior margin of the ramus. Then a second incision was made deeper, and under the submaxillary gland, the gland lifted up until the inferior margin of the ramus could be felt under it. Then the operator had in one mass the submaxillary gland and the lymph glands in the neighborhood. The outer flap was composed of skin, the superficial fascia and platysma. Then the muscle attached to the inner surface of the mandible was severed, and the flap from the margin of the mandible dissected upward, cutting it as close to the mandible as possible without cutting any of the infected tissue, lifting up the mandible so as to carry in the flap every branch of the facial nerve. Using a ribbon retractor, it was quite easy to bring the whole mass up, so as to expose the temporomandibular articulation and disarticulate the mandible. Drainage tubes were placed below, so that the maximum scar is at the point fairly well concealed when the wound has healed.

The impression seems to prevail that these patients after removal of half a mandible are in a very serious condition. This man, as a matter of fact, says he is enjoying himself just as much as ever. If he were to enter a room nobody would notice that the operation had been performed.

After this operation the speaker had the opportunity of having Dr. Wilson, of Cleveland, see the case, and he expressed the opinion that it was not advisable to attempt to make anything to replace the half jaw.

The prognosis in this case Dr. Dean considers to be good. Before the operation he had felt the chances were good, and had urged the man to have it done because of the fact that it had been slow growing, and with the exception of the glands right between the submaxillary gland and the ramus there was no involvement of the lymphatics, so far as he could determine. At operation, search was made for enlarged glands in the neck, and none were discovered.

Specimen of Giant Celled Sarcoma of Jaw.

The second specimen was exhibited by Dr. Dean because of the first one. It was a giant celled sarcoma of the jaw, section of which showed how the growth did not involve the bone. The speaker's conception of tumors of the mandible is that the socalled giant celled sarcoma is the only sarcoma where we are justified in not removing one-half of the mandible. The only other malignant tumor where we are justified in not removing one-half is an epithelioma involving the alveolar process.

DISCUSSION.

DR. GEORGE W. BOOT asked Dr. Dean what he did to control hemorrhage in the first case reported.

DR. DEAN said there was practically no hemorrhage, and what hemorrhage there was could be controlled easily by the use of the hemostat.

DR. JOSEPH C. BECK asked if there was any X-ray picture taken.

Dr. Dean replied that there was, and it was very suggestive of a cyst of the mandible.

Dr. Beck asked if the X-rays had been used therapeutically, to which Dr. Dean replied in the negative. The only use of the X-rays had been to take a picture before operation.

Paper: Pathologic Affections of the Cavernous Sinuses.*

By H. G. Langworthy, M. D.

DISCUSSION.

Dr. L. W. Dean, Iowa City, Iowa, said that in connection with disease of the cavernous sinuses he had had three experiences. The first case was one that came into the University Hospital about three years ago with a septic temperature. There was no exophthalmos. There was optic neuritis on the right side, but the patient had a very bad chronic suppuration of the sphenoid. That was all that was found. Within two days, however, he had a marked exophthalmos, and died about four days after first coming under the speaker's observation. At the time he was first seen he was a walking case with a septic temperature, chills and fever up to 105 degrees, then dropping to normal. Postmortem showed just as typical phlebitis of the cavernous sinus as one ever saw in the lateral sinus, with suppuration of the mastoid. There was a necrotic area of the wall, a dehiscence of the inner wall of the sphenoid, and over this dehiscence there was a necrotic area of the cavernous sinus, with the septic clot. Postmortem made the diagnosis positive.

That case has been of interest to Dr. Dean because there was positively no trace of exophthalmos or edema of the nerve head when the patient was first seen.

The second case was one of arteriovenous aneurism that had been seen by Dr. de Schweinitz, and the diagnosis confirmed by him. Two years ago the speaker ligated the common carotid in that patient, with a fine result. This was a traumatic case, with the pulsating exophthalmos accompanied by terrific pain. By "fine result" he meant that it was fine so far as the pain and slight diminution in size of the tumor were concerned, comparatively speaking, the principal factors in a "fine result" in a case of this kind. This patient is of especial interest to Dr. Dean now because he has a beautiful collateral circulation.

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*See page 560.

About two months ago he went through the literature to find out just how this collateral circulation was established, and talked with Dr. de Schweinitz, who everyone knows has done more work than anyone else in the world on pulsating exophthalmos, and his opinion regarding collateral circulation was not very definite. Dr. Dean had Dr. Prentiss look this second patient over about a month ago, and the conclusion was that the collateral circulation was mostly through the vertebrals. As a matter of fact, he said, it was not a definite opinion, but that was the best he could give.

The third case was one that was reported before the society, and occurred following tonsillectomy many years ago. The patient had a septic temperature coming on after exposure. He ran away from the hospital, exposed himself, and got an infection. When seen by the speaker, his temperature was 105 degrees. He had exophthalmos on the right side. The temperature was typically septic. Optic neuritis was also present on the left side. This patient recovered without a doctor, and came to Chicago afterwards and had the right eye enucleated. Dr. Dean has always thought the case was one of cavernous sinus phlebitis which recovered, but has had no definite way of knowing whether it was that or a sepsis of the orbit.

Dr. Joseph C. Beck said it had been a pleasure to listen to Dr. Langworthy's paper, in contrast to the paper of a similar topic which he read before the Ophthalmological Society, which consisted of a large number of data, and which was absolutely of very little value to the speaker. The paper presented this evening was entirely different. It was practical, and while on a rare subject, yet he was sure that all the members appreciated the work done by the essayist.

The question of intermittent exophthalmos, while perhaps not the subject for discussion, was interesting. Very recently Dr. Beck has had a case of intermittent exophthalmos in which he made an exploratory operation, or rather, was preparing to do an exploratory operation on the orbit for a possible tumor within the orbit. There was, however, nothing definite to show this condition. This patient had been studied carefully by ophthalmologists, with no evidence of any intraocular disease. The condition had no effect whatever on the vision, but it was stated by the physician in charge that every time the patient became excited, nervous and tired, she got an exophthalmos. Dr. Beck never saw it himself, but when the patient was put under the anesthetic, and while he was preparing for operation, he looked around to see the face of the patient, he and all present saw the disproportion of the two eyes. He scrubbed up as quickly as he could and examined the eye; he felt above the inner canthus, and deep in the orbit he felt a mass something like a rubber ball. He, therefore, decided that there probably was a tumor—maybe an angioma—within the orbit, and on the nasal side. Nasal examination was always negative. X-ray examination did not give any hint as to the condition. Incision was made, as would probably be done for a Killian, only further inwards in the orbit. As soon as he came on to this mass, a slight opening was made, and a real gush of pus went all over the face of the patient. This pus was reddish yellow in appearance, like innocuous pus. After wiping out a quantity of this material and exploring further, he found that the entire ethmoid had been destroyed. The orbital periosteum was intact—no erosion—but thickened. In other words, it was a case of infected cyst of the ethmoid, both anterior and posterior, reaching clear back. Outside of the intermittent exophthalmos there was nothing unusual in this case, and he wished to call attention to the fact that one might have suspected in such a case a cavernous sinus condition, as mentioned by the essayist. If he had heard the paper of Dr. Langworthy before he operated, he would have thought his patient had perhaps such a condition of the cavernous sinus. The bacteriologic examination of the pus was pneumococcus, and the subsequent course was that after a middle turbinectomy and ethmoid opening. The patient made an uneventful recovery.

Dr. Langworthy referred to arterial and venous conditions where the carotid artery and cavernous sinus were exposed in the sphenoid sinus. He (Dr. Beck) once had a specimen from Professor Hoove, of Iowa, in which the carotid artery and a portion of the cavernous sinus were free in the lateral wall of this sphenoid sinus.

In regard to the point made of injuring the cavernous sinus in a Gasserian ganglion operation, the speaker had operated one such case, and in severing the posterior root he could very clearly see the cavernous sinus and see how easily one might injure it in that operation.

Dr. Dean, in his discussion, mentioned the ligation of the internal or common carotid artery. Only three days ago the speaker operated on a patient for cancer and resection of the lower jaw and part of the soft palate. He was forced to ligate the common carotid artery. There followed a hemiplegia, with the patient in good condition after operation, and within twelve hours after operation the patient died, apparently just because of his heart giving way. While he recovered consciousness, he could not speak after the operation.

Two months ago he did a similar operation on the lower jaw and part of the upper in a malignant case, doing a temporary compression on the common carotid artery, but it was not correctly done. The forceps were applied too firmly, and on relieving the artery forcep he noticed a distinct compression of the artery, which he feared would necrose, and possibly a fatal secondary hemorrhage ensue. He, therefore, ligated the common carotid, and that patient had the same symptoms—hemiplegia—with death following.

Looking up the literature, he found that twenty-five per cent of cases of ligation of either the internal or common carotid die from just such symptoms, and he wished to say, while this was not a point in discussion, that he would keep away from such procedure in the future.

He wished to call attention to the reaching of the cavernous sinus very easily by a procedure first proposed by him for reaching the hypophysis, namely, through the antrum, being able to reach the opposite side. For instance, in operating on the right cavernous sinus he would go by way of the left antrum of Highmore, in through the sphenoid, and then on to the cavernous sinus on the opposite side. It would certainly be an easy method by which we could reach it. He has done it on a cadaver a number of times, and in two cases on the living, in operating on the hypophysis.

Dr. Harry Kahn referred to a case which a general practitioner operated some few years ago for the removal of tonsils and adenoids, but did not get the adenoids out completely. Dr. Kahn operated on the adenoids under a general anesthetic, and sent the patient to bed. About twelve hours after operation the right eye began to get larger, and finally there was a proptosis. The eye was greatly swollen and looked like a plum. The patient had a septic temperature for two or three weeks, and then, by expectant treatment, the whole thing disappeared. The speaker did not know whether that was a cavernous sinus affair or not. He was unable to make a definite diagnosis. Dr. Frank saw the case with him. There was no pus in the nose, and no evidence of anything except the proptosis, septic temperature, and meningeal symptoms evidenced by headache and vomiting. Treatment consisted of urotropin, hot applications, and so on, and the patient entirely recovered without any loss of sight or disturbance of any sort.

He felt it was worth while to report such a case following an adenoid operation.

Dr. J. Holinger was very much surprised to hear nothing in the paper, nor in the discussion, about the progression of the thrombosis to the cavernous sinus from the lateral sinus in mastoiditis and inflammation of the ear. We know that the progression of the thrombosis to the cavernous sinus and other sinuses of the base of the brain is one of the most frequent causes of death in cases of thrombophlebitis of the lateral sinus. Certainly he would think that this would be at least as important as the eye symptoms, exophthalmos, and so forth. The progression of the thrombophlebitis in that direction is discussed to a great extent by any number of ear men, and especially Leutert, many years ago, in his strong plea against ligation of the jugular vein in cases of thrombosis of the lateral sinus. He made the statement that undoubtedly most of the cases of progression of thrombophlebitis along the cavernous sinus from the lateral sinus might be avoided if (1) the lateral sinus were not handled so much in the operation, and (2) if the jugular vein were not ligated. He advised simply to lay bare the lateral sinus to as large an extent as possible, but otherwise leave it alone. Not even the splitting of the sinus is advocated.

This is the process which brings ear surgeons most frequently in contact with diseases of the cavernous sinus, and we have learned to look at any reliable sign of thrombosis of the cavernous sinus as a pretty sure signal of a fatal termination of a case of sinus thrombosis.

DR. GEORGE W. BOOT had had one case of thrombosis of the cavernous sinus in a young civil engineer, who was working out in stormy weather. He had a furuncle on the side of his nose and was brought to St. Francis' Hospital in Evanston with a well-marked thrombosis of the left side, which very shortly—in the course of twenty-four hours—extended to the right side, so that the eyeball protruded a great deal, with a great deal of ecchymosis. This man had meningeal symptoms, and in two or three days died of meningitis.

Shortly after this he saw a boy of about eleven years who had an exophthalmos of the left eye. He had a chronic suppurative otitis media on the left side. In this case those who saw the patient thought he had the condition referred to by Dr. Holinger, namely, an extension of the thrombosis from the lateral sinus to the cavernous sinus. Accordingly, a radical mastoid operation was performed, but failed to relieve the condition. Shortly after it became evident that the trouble was not in the sinus, but was a sarcoma within the orbit.

Dr. Boot saw a third case not long ago, in which the question of thrombosis of the cavernous sinus was considered, but finally ruled out. The final diagnosis was that of inflammation of Tenon's capsule, although there was no tenderness of the globe on pressure.

DR. LANGWORTHY, in closing the discussion, said there was not a great deal to be added to what he had already said in his paper. In a single evening it was quite impossible to cover all of the points which might come up in a discussion of the cavernous sinus, and especially its relation to other parts of the head. While he had been interested in this subject for a long time, it is very difficult and almost impossible to get material to work with, and our knowledge of conditions must be accumulated in the way brought out in the discussion. The object of his paper was to systematize the cavernous sinus and treat it as a separate organ, if possible, and to classify the pathologic effects, which task had not been easy. He realized that it was not a practical field for labor, but with the attention that the pituitary body is receiving, and will continue to receive, it seemed to him that that would probably throw more light on the cavernous sinus and the structures adjacent to the sphenoid bone.

Stereoscopic X-ray plates, for instance, ought to be of help in clearing up a great many possible affections of this sinus. He has seen some of the most beautiful plates in Dr. Beck's clinic, and they have certainly been most instructive, and have suggested the possibility of obtaining definite knowledge beforehand in certain affections, and when one stereoscopically can look into a head, as it were, and see the lines of the bones, just as though it were a glass bowl, it is certainly a wonderful sight. There is probably no limit to the heights that can be reached along these lines.

Dr. Dean's experience with several cases in some ways is unique. The speaker doubted whether many present had seen many cases of thrombosis. Some of Dr. Dean's reports had been incorporated in the tables which accompanied the paper.

Regarding Dr. Beck's remark, especially relating to the paper read before the Ophthalmological Society, that was a paper which dealt purely with thrombosis and its relation to the future; that is, the matter of possible early diagnosis, and the various ways of reaching the cavernous sinus, all of which, however, have still to be worked out. Dr. Beck's method is certainly as practical as any. Dr. Langworthy had attempted to work this out himself, and it certainly seems that some method of going upward through the nose might be one of the first things thought of in dealing with thrombosis.

Regarding Dr. Kahn's interesting case, he hardly felt like commenting on it, from the meager reports which he gave. It was evidently one in which a diagnosis would be very difficult, and he would want to have more complete information.

With reference to Dr. Holinger's remarks, he wished again to say that the paper had to deal with the matter of classification, and was not an attempt to cover the single subject of thrombosis, which would require a good many pages.

Whether anything practical would come out of the study of the cavernous sinus or not, he did not know, but it seems to him that in dealing with some of the cases, a few of them would come under the surgeon's care, and possibly be relieved by operation.

Paper: Two Severe Cases of Ulceromembranous Angina.*

BY IRA FRANK, M. D.

DISCUSSION.

DR. ROBERT SONNENSCHEIN thought the paper of Dr. Frank very interesting and valuable for many reasons, but particularly in pointing out two important facts. First, the marked clinical contrast between cases of socalled Vincent's angina, and, second, the fact that fatalities may occur in this disease. To judge from the literature, one would imagine that these cases are practically all favorable in their prognosis. It was his privilege to see at least once both of the cases mentioned in the paper. The first one was already in the stage of healing when he saw it, but the second case was seen one day when Dr. Frank was out of the city, at which time an attempt was made to introduce food into the stomach, owing to the fact that the patient was unable to swallow in the ordinary way, by passing a very fine tube-like catheter through the pharynx. This failed, however, owing to the fact that the patient immediately began to complain of severe pain.

Last spring Dr. Sonnenschein saw two cases in the Skin and Venereal Ward at the Cook County Hospital, both men, one white and one colored, and both died. This disease may and oftentimes does end in death.

Just one point further that is not entirely relevant to the subject, namely, the name of this disease—Vincent's angina. It seems to him that in justice to a man who has done the work the name ought to be changed. Arthur Meyer wrote a large monograph on this subject, and showed that it was Plaut who described the organisms a year and a half before Vincent. Owing to the fact that the latter got his name into the literature, the disease has been called after him.

DR. ALFRED LEWY reported, from a very limited experience with the milder form of ulceromembranous or Vincent's angina, that sodium perborate has been very efficacious in its treatment. He has given up the trichloracetic acid application, simply cleansing the ulcer with peroxid and swabbing, and using a perborate gargle, and application of perborate of sodium powder, with great relief, so far as discomfort was concerned, and great help in rapid healing.

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*See page 637.

Dr. George W. Boot said that the two cases referred to by Dr. Sonnenschein at the County Hospital he had had the pleasure of seeing twice. A third case he had expected to show to the society this evening—a colored woman of about sixty years, who entered the County Hospital with very marked ulceration, with formation of Vincent's membrane over the posterior brim of the pharynx, tonsil and pillars of the pharynx. Smears gave the spirillum and fusiform bacillus. The patient failed to improve under local antiseptic measures. Wassermann examination positive. She was put on antisyphilitic treatment. The ulceration healed, but with a stenosis of the nasopharynx. There was only an opening of perhaps one-half centimeter in diameter between the oropharynx and nasopharynx. He is at present attempting to restore the opening, following the method of Rose, of putting a stitch through this small opening remaining and bringing it out at the side, about where the normal location of the side of the pharynx should be, tying the silk stitch and leaving it in for a considerable time, hoping the epithelium will line the canal. Then he expects to divide the tissue.

Vincent's angina, socalled, it seems to him is hardly a distinct entity, in that the organisms of Vincent's angina are present in so many different conditions in the mouth, without making a distinct disease. They are found in carious teeth, and he has seen smears from diphtheria patients, from the false membrane, give large numbers of the spirillum and bacillus, which were mistaken for the fusiform. So that he thinks we can consider Vincent's angina as primarily a deposit on another process; that is, in any ulcerative condition in the mouth you can get the same condition as in Vincent's angina, without it being a distinct disease.

Dr. Joseph C. Beck said that Dr. Boot's discussion reminded him of a case he saw in consultation, in which there was a fatal result in an ulceromembranous angina. When he saw the case the history was given that the specific treatment was applied, namely, painting with ten per cent carbolic acid solution, daily, probably for a week or so. He could readily see where the use of that remedy might enhance the action of the infective organism by lessening the vitality of the tissues and producing a local necrosis, and then subsequently this ulceration. He would like to know whether there was anything like that used in the cases reported.

Second, he wished to call attention to the work of Professor Siebenmann on ulcerative sore throat from the leptothrix. Four cases of that same disease, ending fatally, were reported by Brown Kelly, of Glasgow. Was anythink like that looked for in the cases of Dr. Frank? Did Dr. Frank make any report on the bacteriologic or smear examination?

This condition described by Dr. Frank is known as Plaut-Vincent's angina, and it is also known that we find the leptothrix in the mouth, especially when it is in an unhygienic condition; in cases of low vitality of tissues, and general debility, the leptothrix will thrive to the extent of producing marked ulceration. It is well worth while to bear that in mind in treating Vincent's angina.

DR. FRANK, in closing the discussion, said, in answer to Dr. Beck, that a smear and culture were taken, but nothing particular was found.

Answering Dr. Lewy, he had never tried the remedy referred to.

Salvarsan has been used in cases of this character, both locally and subcutaneously, but he did not use it, for the reason that it is too expensive now.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Regular Meeting, Held February 29, 1916.

DR. OTIS H. MACLAY, THE PRESIDENT, IN THE CHAIR.

Histologic Specimen of Tumor Removed From Larynx, Together With Remarks on Direct and Indirect Laryngoscopy.

DR. NORVAL H. PIERCE reported the case of a man who came to the Illinois Eye an Ear Infirmary complaining of hoarseness. There was no pain. Dr. Schoolman made a diagnosis of tumor of the larynx, situated on the left vocal cord, very close to the anterior commissure. Direct laryngoscopy with the most recent type of apparatus was tried, but the speaker was unable to see the tumor because of its situation and the thick, short neck of the patient. As much force as he regarded as justified, both internally and externally, was used to bring the tumor into sight, but without avail. The man bore all this as well as could be expected, although complaining very bitterly about the pain and discomfort, especially as he had pyorrhea of all the teeth. In contradistinction to this maneuver, it was very brilliantly demonstrated that indirect laryngoscopy, in his particular case, at least, was very much to be preferred. The tumor was very easily removed on the first serious attempt, and there has been no recurrence. Histologically, it proved to be a fibrolipoma. Those who had examined the man could see that the place of attachment of the tumor is hardly to be discerned under the laryngoscope. He brought up the point regarding direct and indirect laryngoscopy to accentuate the fact that each method has its own particular field of application.

Suppurative Meningitis, Probably of Syphilitic Origin.

DR. NORVAL H. PIERCE: The patient was a male, laborer, aged fifty years. No tuberculosis or cancer in family history. Chancre fifteen years ago, but no secondary signs of syphilis developed. Past history showed measles at seven, followed by corneal ulcer; also corneal ulcer (right) four years ago. No other illnesses. Present complaint began last April with pain behind right ear, accompanied by discharge. In August the ear became painful. The tympanic membrane apparently ruptured, and discharged pus for several days. Last September he began to have headaches, and in November he quit work. He had attacks of dizziness at short intervals. On December 5th he fell to the floor three times in the morning. From December 15th to 21st complained of frequent attacks of dizziness and headache. This continued, accompanied by loss of appetite and also loss of weight. As to his condition when he entered the clinic, he complained of slight headache and pain in the right ear. Paracentesis was performed by Dr. Lewy. He was admitted to the hospital on the 13th of December. Another paracentesis was performed by Dr. Smith, and followed by a slight purulent discharge, which stopped by February 4th. Was sent home, but returned to the hospital complaining of headache and dizziness. At this time examination of the right ear showed profuse purulent discharge; left, chronic catarrhal otitis media. On January 29th, white blood count, 12,300; hemoglobin, eighty per cent; albumin in urine; no sugar or casts. Hearing tests: Right ear, voice not heard; osseous and aerial conduction not heard. Left ear, whisper, fourteen feet; osseous conduction of an a^1 of five seconds; aerial conduction, fifteen seconds. Low limit for left ear, $G._2$ Weber test, to left, fifteen seconds. Nystagmus marked to left. Rotation: Eight seconds' nystagmus from diseased ear, and thirty seconds from left side. Cold irrigation of left, nystagmus to right on looking ahead. Pointing test showed deviation to left. No response to cold or heat in right ear. February 2, 1916, complained of severe pain in occiput and neck. Very dizzy. Cerebration low. White blood corpuscles, 6,400. February 3, 1916, Wassermann negative. Urine showed albumin, two per cent sugar; casts absent. He was given iodid of potassium at this time, which was continued practically up to the time of death. Chest and abdomen negative. Blood pressure, systolic, 140; diastolic, 70. Temperature, 100°; in morning, 99°. At no time was the temperature above 100°. Cerebration still low.

Examination of the reflexes showed pupils reacted to light and accommodation slowly. Knee jerks weak. Babinski negative. Muscular strength slightly reduced on left side. Fundus examination unsatisfactory because of corneal opacity.

The relatives of the patient refused all operative procedures, but lumbar puncture was made on February 5th. Fluid under pressure. The opalescent spinal fluid showed albumin, 2.2 per cent. White blood cells, 787 per cubic millimeter. Bacteriologic content not reported.

February 6, 1916, slight stiffness of the neck. Slight Kernig on both sides. Marked spontaneous nystagmus to both sides—horizontal and slightly rotatory.

On February 8th the condition was unchanged. The patient went into deep coma and died on the 9th, at 4 a. m. On autopsy a very interesting temporal bone was found. A large mass of tissue, having the consistency of rubber, entirely occupied the jugular bulb. Its character could not be determined without microscopic examination. It might be an old organized clot. Its appearance would suggest that as the most probable thing. But there were no symptoms of sepsis at any time while he was under the speaker's observation. It might possibly be an organized gumma, but there were no symptoms of interference with cerebral circulation, when he was first seen at least, and we would expect that to be present if it were a gumma. However, that could only be ascertained by microscopic examination. Unfortunately, the cochlea was sawed through at autopsy, but it showed nothing macroscopically except that it was pretty well filled up with rather a homogeneous material, which probably resulted from the suppurative labyrinthitis. The temporal bone was perforated on its roof, and this condition again suggested syphilis, as the perforation went right straight through the upper portion of the bone into the sigmoid groove, whereas the perforation of the mastoid was posterior. This peculiar destruction of the bone would suggest a syphilitic process. It was hardly a well defined osteomyelitis. The lateral sinus contained no clot. There was fluid blood in it, which again interfered with almost any theory one might have regarding the growth in the jugular bulb.

The next point of interest was that in a case of suppurative meningitis the blood count might be as low as 6,200, with rather a lower fever—subfebrile—and absence of stiff neck. He complained of headache toward the last, but it was not marked. He had none of the classical symptoms of a meningitis, and, in fact, the diagnosis made at the time before death was that there was some cerebral complication. The brain was preserved, cut, and shows very beautifully the manner in which the pus traveled along the arachnoid spaces. It shows how impossible it is to drain a suppurative meningitis. (Dr. Pierce showed the specimen.)

DISCUSSION.

DR. GEORGE E. SHAMBAUGH thought that the second case reported by Dr. Pierce permitted of some speculation as to the diagnosis. It seemed to him quite unlikely that a patient could be examined, even in a superficial way, and a suppuration of the labyrinth overlooked; in fact, not even the suppurative otitis media recognized. As a matter of fact, it may be very easy to overlook a chronic suppurative otitis media with a fistula in the region of Shrapnell's membrane. Such cases often give no history of discharge, and all that one detects is a dry scab, which is not easily removed and might very readily be mistaken for a little flake of cerumen. It is only after dislodging this scab that the fistula can be discovered.

It seemed to the speaker that it was extremely unlikely that a patient would develop the complication of suppurative labyrinthitis secondary to an otitis media, and not give very accurate symptoms pointing toward this lesion. The onset of a suppurative labyrinthitis is a tremendous shock to an individual, and is associated with very marked symptoms. In view of this fact, the speaker wondered whether the intracranial trouble in this case did not have its origin in syphilitic infection, as suggested by Dr. Pierce, and whether the otitis media, which was recognized the second time the patient was seen, was not an independent affair quite distinct from the fatal complication. The dead labyrinth has to be accounted for, but, as we all know, this is not such an uncommon occurrence, independent of any suppurative otitis media.

DR. J. HOLINGER thought the tumor seen in the bone had the typical consistency of a gumma.

DR. PIERCE said that the specimen had been in formalin for some time; it was not so gummy when first removed.

Dr. Holinger said that the inner plate of the bone was rough and uneven—another peculiarity found in syphilis. The whole course of the labyrinth affection would also suggest this etiology. He thought the perforations from the sinuses, in different directions, gave a clear explanation that on account of the gumma in the bulb of the jugular vein the flow of blood was interfered with, and so was the collateral circulation.

Dr. George W. Boot did not agree with Dr. Holinger. It looked to him very much like an old organized thrombus in the jugular vein just below the bulb, because the process extended below the jugular bulb. The opening in the bone, aside from the perforation, caused by the disease, was an anomaly. Syphilitic meningitis is not apt to be suppurative.

Dr. Joseph C. Beck was inclined to agree with Dr. Boot's diagnosis. He thought it was an old chronic suppuration. The patient being so long ill, his vitality would have been reduced to some extent, also his resistance, and there would be no possibility of a high blood count. He would like to know what the differential count showed.

By making a Wassermann test of the spinal fluid we have a most definite diagnosis of malignant syphilis. That should be done for differential diagnosis.

He saw the perforation in the specimen of the temporal bone, and the tumor, but did not examine it; however, he thought it was probably due to an old organized thrombus in the jugular bulb.

Dr. J. R. Fletcher wanted to call to Dr. Pierce's attention the fact that there was a break through the jugular bulb to the posterior fossa. He did not know whether Dr. Pierce had noticed this or not. That would make the condition somewhat of a problem. It seemed to him that it was an old thrombus, which caused this bone destruction during its active period. Meningitis or cerebellar abscess should, however, have developed at this time.

Dr. Pierce, in closing the discussion, said that the condition other than the meningitis did not look to him like a simple inflammatory one.

The orders were left to make a Wassermann on the spinal fluid, but through some mistake it was omitted.

There was no tenderness or swelling over the mastoid to indicate active inflammation within the mastoid.

He presented the specimen this evening simply as it came from the dead body. His next step will be to investigate the interior of the mastoid, and find whether there was a suppurative process within the mastoid.

Personally, his diagnosis as to the ear disease was syphilis, but, of course, this would not explain the whole picture. He died clearly of a frank, well defined, suppurative meningitis. But back of that there probably was a syphilitic process. There might be further data acquired when the labyrinth was sectioned.

Dr. Shambaugh thought the diagnosis might have been made when the patient first came to the hospital. If he had had any labyrinthitis before, or had it at that time, the speaker was not prepared to say, but at least, when he came to the hospital the first time, it was in a quiescent state, so that there were no symptoms of it. He gave no history of previous spells of dizziness, so that in the actual working of the clinic he was passed through with the diagnosis of an external otitis. The external auditory canal was very much swollen, and it was very difficult to get a view of the tympanic membrane. The whole thing will be largely cleared up by further investigation.

Giant Formation in Larynx Accompanying Hypophyseal Trouble.

Dr. Joseph C. Beck presented a man, sixty-five years of age, who came to him only a few days before, with laryngeal difficulty, which affected both speech and breathing. On examination, a large mass, about the size of the speaker's thumb, was found, which was soft, and fell into the larynx. The man's physiognomy has changed in the last year. The lips became very large; the tongue also became very large, and his head and features have enlarged. X-ray examination showed definite destruction of the sella turcica. The man had hemianopsia in one eye. The other eye was blind, but this was due to smallpox scars on the cornea.

The interesting point in this case was the occurrence of a laryngeal trouble in connection with hypophyseal disturbance. Such cases of giant formation in the larynx have not been described in the literature, so far as Dr. Beck knew. The patient was suspended that day, and the mass referred to could be moved. It looked to Dr. Beck like a hypertrophy of the mucous membrane, with relaxation.

Complete Destruction of Nose.

Dr. George W. Boot showed a case, which the members had seen before, of destruction of the nose, the septum and all the turbinates, in a man who had received an injury several years ago, and had been treated for syphilis. The man attributed the loss of the nose to the treatment, but it was probably due to syphilis. A year ago he had a strongly positive Wassermann reaction. He refused to have a new nose made. Vision was all he wanted improved, but that was impossible.

The interesting part of the case, aside from the destruction of the nose, was the manner in which the action of the soft palate could be seen when the patient spoke.

Lupus of the Nose.

Dr. George W. Boot: The second case came to the clinic at Rush the afternoon of the meeting. He was only sixteen years old; of Italian parentage, but born in England. The trouble had lasted for three days. He was treated in a London hospital, as he says, by the Finsen light, and under this treatment for nine months the condition of the nose cleared up entirely. Then he came to this country a year and a half ago, and the nasal trouble has recurred. The diagnosis made by one of the men in the skin clinic was tertiary lues; the speaker made one of lupus. The boy had a positive Wassermann reaction, two plus, but denied any knowledge of any venereal infection.

Probable Gumma of Frontal Sinuses.

Dr. Robert Sonnenschein reported the case of a man, aged twenty-nine years, who had a chancre in May, 1915, with general adenopathy. Shortly thereafter he had a very profuse, large maculopapular eruption. He developed severe pain and headaches. He was immediately put on inunctions of mercury and large doses of potassium iodid. He was also given nine intravenous injections of neosalvarsan. He had ninety mercurial injections altogether. The patient developed very severe pain in the region of the left frontal sinus; later, also in the right, and at present there is still pain in the right sinus, which is especially severe at night. Morphin has very little effect on the pain. At no time was any pus found in the nose, although the middle meatus was opened and suction applied. Externally, nothing definite felt or seen, but tenderness over the frontal sinus, especially at the external angle. X-ray showed a peculiar circumscribed area in the extreme external end of the right frontal sinus. Just what that shadow means the roentgenologist could not say. Gumma had been suggested. Dr. Pierce suggested a possibility of a neurologic condition, and advised that the temperature be taken and a leucocyte count made. Any suggestions as to diagnosis and treatment would be gratefully received.

Paper: Surgical Anatomy of Frontal Sinus, With Demonstration of Anatomic Specimens.*

By George E. Shambaugh, M. D.

DISCUSSION.

Dr. Charles M. Robertson said that operation on the frontal sinus, by either the external or internal method, should be determined by skiagraph, and he agreed thoroughly with Dr. Shambaugh regarding the external operation. He has seen cases in which the ostium opened on the other side. For instance, the right frontal sinus had no opening on the right side of the nose, but on the left side, separately from the left frontal sinus. In nearly all of these cases a very small amount of drainage should be sufficient to cure the case, because if we have a small amount of cavity we have a small amount of pus, and if we have a small cavity, the cavity is filled very easily and the pus forced down into the nasofrontal duct. Those are the cases that get well of themselves. The cases that do not resolve are those in which the frontal sinus is extensive, as Dr. Shambaugh said, extending back over the orbit or out to the zygoma. He saw one cadaver in which the frontal sinus extended clear out into the zygomatic process and communicated with the cells of the mastoid through the zygoma. This was a pneumatic skull absolutely; the two mastoid processes communicated through the occipital bone, and this, too, was the only case that he had ever seen in which the frontal sinuses were above a very large and numerous set of ethmoidal cells that extended clear around the orbit, so that there was a partition between the ethmoidal cells and the antrum of Highmore. If anyone had attempted to cure an empyema in that case by either external or internal operation, he certainly would not have succeeded. It would have been absolutely impossible to have gotten drainage from all of those cells.

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*See page 707.

At present, the internal operation is the one of selection. The ethmoidal cells that push up usually open into the nose by separate openings, although, as Dr. Shambaugh said, some of these ethmoidal cells have one frontal sinus opening into them. The whole business is an ethmoidal cell pushed up into the frontal sinus; the antrum of Highmore is an ethmoidal cell pushed down into the superior maxillary bone. They are a combination of cells that are thrown in to give the skull less density, less weight, and also for a resonating cavity, and in each case the cells differ. Dr. J. C. Beck several years ago said that these skulls were usually bilateral in their configuration. We find that is not so in the frontal sinus, and very often not so in the ethmoidal cells, one sinus being very large, extending out to the absolute edge of the zygoma, and the other one very small, or no cell at all.

The matter of external operation with deformity has never frightened him as much as some others, because in doing it he has always left a broad bridge, in which the deformity is absolutely at a minimum. Experience has taught him that very many of these ostia rather hug the cribriform plate, more than was indicated by Dr. Shambaugh. In fact, in nearly all the cases that he probes, the direction is more upward, forward and inward than outward.

Dr. Otto T. Freer thinks it helps the anatomic conception of the frontal sinus and of its relation to the ethmoid cells to appreciate that the sinus itself is merely an ethmoid cell that has invaded the substance of the frontal bone, as the other ethmoid cells are inclined to do, in the manner, as Vacher says, of a bubble blown into glass. The penetration of other ethmoid cells into the frontal bone is made evident by looking at the border of the ethmoid notch of the frontal bone where are seen the depressions or foveolæ which complete the uppermost cells of the lateral masses of the ethmoid bone. In many specimens it may be seen that the foremost of these ethmoidal foveolæ has entered into the frontal bone to form a large cell lying just posterior to the frontal sinus, having the same general form as the sinus, and often extending laterally beyond it, such cell being called a "bulla frontalis." These anatomic facts are clearly shown in Figures 1 and 2 in Dr. Freer's recent article, "The Frontal Sinus: Opening It Through the Nose."

The structural features mentioned by Dr. Shambaugh have definite anatomic designations which Dr. Freer wished to see in general use, in order to produce a clearer mutual understanding in discussing the intricate anatomy of the frontal sinus region.

Dr. Shambaugh spoke of the lower outlet of the nasofrontal duct as being found either in the infundibulum or in front of it, under the anterior end of the middle turbinate. In the first case the outlet is called an "ostium infundibulare," in the second an "ostium recessuale," as it opens directly into the hollow extending upward towards the frontal sinus under the middle turbinate, a space called the "recessus frontalis" by Killian.

The prolongation of the frontal sinus backward alongside the cribriform plate, mentioned by Dr. Shambaugh, is called a "recessus paracribrosus" by L. Onodi. Where the cribriform plate and the olfactory fossa, of which this plate forms the floor, project forward enough to create a bulge in the posterior wall of the frontal sinus, the projection into the sinus so formed is called a "torus olfactorius" by L. Onodi. The wall of such a torus not only projects strongly into the sinus on the inner posterior part of the circumference of the sinus outlet (ostium frontale), but this wall is often very thin, so that Herbert Tilley, in describing the intranasal opening of the frontal sinus, warns against enlargement of the ostium inwardly, because the cranial cavity might be opened here.

Very small frontal sinuses, mentioned by Dr. Shambaugh, are common in women with smooth foreheads lacking frontal eminences. Such sinuses may be mere slit-like cavities lying just above the brow. To such faces, as a group, Dr. John Gordon Wilson has given the name of "monkey-face type." In spite of their small size, little sinuses, if chronically suppurating, may cause anterior orbital abscesses by creating caries of the floor of the sinus.

Hajek calls attention in his work on the anatomy of the sinuses to the difficulties met with in probing the frontal sinus, and shows that the false direction apt to be given the probe is mainly due to the lower border of the middle turbinate, which directs the probe too far outward, involving danger of pushing a stiff probe into the orbit. Where the probe, therefore, will not readily slip up into the sinus, the middle turbinate should be resected anteriorly before further attempts at probing are made, care being taken in all probing never to use force.

The prevalent idea that chronic frontal sinus suppuration cannot get well unless the mucous membrane lining of the sinus be curetted away is as mistaken in regard to the frontal sinus as it is in regard to the maxillary antrum. Only rarely, where caries exists, is the mucosa so degenerated that it cannot recover its normal state, if good intranasal ventilation and drainage be secured for the sinus. This is the view of Max Halle, the most experienced and radical of the intranasal frontal sinus operators. Curettage of a noncollapsible cavity with rigid bony walls cannot have the beneficial results offered by curettage of a cavity whose walls are composed of soft parts. In the latter case, curettage establishes healthy granulations which enable the walls of the collapsing cavity to grow together, obliterating it. In a bony cavity no such collapse can take place, and curettage replaces epithelium-lined mucous membrane, capable of recovery, with a granulating surface, which merely sprouts exuberantly and continues to suppurate.

Dr. George W. Boot showed a specimen with a very large frontal sinus divided by numerous septa that were cut off in making the preparation. The one peculiarity of this sinus was that while most of the septa ran in an anteroposterior direction, there was still another running in a transverse direction, and behind it a large cell, in which he could place a hook.

Dr. Norval H. Pierce reported a case, of which he was reminded by one of the specimens. A young woman had an intranasal operation. The case was characterized by great pain for some length of time. The intranasal operation gave little or no relief, and then she was operated externally. The pain continued, but abated for some time, when she came to the speaker's notice with a swelling over the left lateral orbital region, fluctuating temperature, and he advised external operation. The wound, which had been very small, was enlarged, and he opened into an abscess cavity at the extreme outer portion of a frontal cavity that resembled the one he referred to, shown by Dr. Shambaugh, to a nicety. The septum had not been removed in operating. The mesial portion of the cavity was quite normal, except that the internal wall was missing, leaving the dura exposed. This lateral portion (indicating) was filled with granulations, and when this septum was taken away, as the speaker did in his operation, and the granulation tissue curetted out, she made a favorable recovery.

Paper: The Oral Cavity in Relation to Ear, Nose and Throat.*

By T. E. Carmody, M. D.

DISCUSSION.

Dr. J. R. Fletcher thought the paper illustrated very forcibly how much we fail to see. We are very liable to neglect the mouth. He recalled a case seen some time ago in his clinic. The patient complained of severe pain in the throat. Examination by several clinicians had revealed nothing. The throat was perfectly normal, but a large abscess had formed around the roots of the posterior superior molar on the right side. Opening that abscess relieved the throat pain. Frequently, in Dr. Fletcher's experience, when the cause of complaint cannot be found elsewhere, he finds it in the mouth. Another case, referred by a dentist, came to his mind. All of the teeth had been extracted years ago. A fistula developed at the side of the upper left middle incisor, which seemed by probing to come from the antrum. At least the last of four dentists to whom the lady had been thought so. Upon examination there seemed to be a large exostosis protruding into the nose from the floor. He thought this might be an unerupted tooth which, upon operation, proved to be the fact. The fistula was a communication between the nose and mouth, around the tooth root.

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*See page 682.

Dr. Fletcher thought the subject timely and exceedingly interesting.

Dr. P. J. H. Farrell said he had heard one of our noted neurologists read a paper upon neuralgia, and the speaker took exception to the statement that in his very wide experience he had never seen a case that could be diagnosed as neuralgia resulting from any condition of the teeth. Dr. Farrell instanced a case where there was an impacted third molar that apparently was the cause of the neuralgia. The patient had this unerupted third molar removed, and was entirely relieved of a very serious neuralgia for a period of three or four months. The neurologist said he did not care—that he had seen some thousands of cases—and to Dr. Farrell's disappointment he saw this case in a few months with a return of his neuralgia as severe as ever.

The speaker had had a number of cases since that time which were similar, and he has come to believe that if the diagnosis is made of neuralgia—a typical tic douloureux—the teeth may be eliminated as causative factors. There is a difference between neuralgia and the pain from an abscess. The dental profession is in the habit of calling any pain in the gums, jaws or teeth "neuralgia," nowadays. Neurologists all agree, the speaker believes, in eliminating the teeth as a cause of neuralgia.

Dr. Joseph C. Beck said that probably Dr. Carmody was able to speak so authoritatively on the subject because he had practiced dentistry before he became an otolaryngologist, and consequently had perhaps seen some of his own mistakes, or those of others, which had been shown so beautifully in the skiagrams. It has been the speaker's misfortune not to get the cooperation of dentists in doing this work. He has had considerable difficulty in trying to exclude a nasal or sinus condition, believing it was caused by the teeth, and asking the dentist to make an examination. The dentist would say that he had been taking care of the teeth, and that there was nothing wrong, sending the patient back to Dr. Beck. Finally, after the patient had put up with this for some time, he would consult another dentist, perhaps at Dr. Beck's instigation, and find just such conditions as those brought out by the essayist.

The speaker said something about every hospital, or perhaps every number of men practicing together having a dentist for their own cooperation. That is Dr. Beck's practice, which he adopted on account of the difficulty experienced.

Dr. Beck cited a case of a young woman who had a nosebleed that he could not stop. He suspected the teeth, sent her to a dentist, who said the teeth were all right. A skiagram showed definite disease. The patient was sent back to the dentist, who told her that what she needed was a good nose doctor. However, there was a dead root held by a small bridge, and not only that, but a sequestrum above that; the superior maxilla had a fistula leading to the middle of the nose, near the sinus, which was the cause of the nosebleed from suppuration.

Another point he wished to make was with regard to the X-rays. The films that are taken by dentists in order to show a clear picture of the absorption of bone in suppuration about apical infections do not assist him as much as does a dental picture that has been recommended by Eisen—who was killed in Milwaukee recently—who made X-ray pictures of half the side of the face to show the antrum and both jaws. Pictures taken in this way give a very good illustration of these diseases in connection with antrum trouble, and the speaker believes that that is the way these skiagrams help in diagnosis.

Dr. Beck was exceedingly glad to have heard Dr. Carmody's paper.

Dr. S. A. Friedberg asked Dr. Carmody whether any other treatment than extraction was of benefit in cases of small abscesses.

Dr. N. Schoolman asked Dr. Carmody whether the finding of teeth in the antrum of Highmore, especially in the case reported as subsequently being found to be a sarcoma, would be conceded as a dermoid cyst, or whether these teeth are found in any other way.

Dr. Charles M. Robertson mentioned a condition of the mouth that is of interest to nose and throat men, and has considerable connection with the teeth, namely, the subject of leukoplakia. He wished Dr. Carmody would say something about the occurrence of leukoplakia, as regards teeth, in which carious or roughened teeth are the primal cause of the leukoplakia. We are interested in leukoplakia for the reason that it breaks down into epithelioma, and from epithelioma into carcinoma. Dr. Robertson has seen numerous cases of cancer of the tongue, of the cheek and of the back part of the throat that were directly caused by rough or carious teeth, either from a bad plate that is anchored on floating teeth, or from pyorrhea teeth that are ragged-edged and irritate the mouth. Leukoplakia used to be considered as a disease accompanying irritation in the shape of smoking and chewing of tobacco. That, in Dr. Robertson's opinion, to a certain extent, is a mistaken idea. He believes that the teeth were the real cause of the leukoplakia, and not the irritation caused by the fumes of tobacco or the chewing of tobacco, as in nearly all these cases the patients were excessive chewers or smokers and also had ragged, rough teeth, with pyorrhea pockets.

The essayist did not bring out very strongly the idea of stretching the palate for the straightening of the septum. In nasal surgery, as it is practiced today, submucous operations are produced on children of tender years, which septums could probably be straightened by the broadening of the palate, as that brings the septum down, whereas it was a V-shape before, and this elongates the vertical dimensions of the septum, pulling it into a straight plane.

DR. H. KAHN asked Dr. Carmody at how late an age one may begin to broaden the jaw in septal cases.

DR. CARMODY, in closing the discussion, said that Dr. Fletcher spoke of extracting all the teeth, and Dr. Farrell also spoke of the extraction of teeth for neuralgia. The cases of real tic douloureux that we see very frequently have had all of the teeth extracted, and it has been found that in those cases the pain is usually located in one tooth. The patient will point out which tooth it is in. As soon as the tooth is extracted, the patient will locate the pain in the next tooth, and keep on until they are all extracted. What Dr. Farrell said is true—those cases do not clear up, if they are true neuralgias, by extraction of the teeth alone. If they are treated generally and brought to a good physical condition, the neuralgic condition will probably disappear, but not because of the extraction of the teeth.

Dr. Beck spoke of his experience, and it has been the speaker's also, only probably not to quite so great an extent, because of the dentists with whom he is associated all know that he was a dentist, and when he tells them what to do for a case, they generally do it. However, he has had the very experience Dr. Beck related. Dr. Beck referred to the taking of pictures, but Dr. Carmody did not know exactly what plates he referred to. However, he has found that the plates, so far as the jaws and teeth are concerned, are unsatisfactory. They do not give the idea of a root very well. They, however, give a very general idea of the sinuses and jaws.

Dr. Friedberg asked whether extraction alone is necessary. In many of the cases, that is all that is necessary, because cavities will drain very well. In others, however, it is necessary to curette the bone, because while we have not a true necrosis there, as we understand it—death of the bone en masse—we have a caries of the bone, and the little fine particles have to be taken out.

Dr. Friedberg asked if every tooth in which there was an abscess should be extracted. Dr. Carmody said that in many cases there is quite an abscess cavity in the tooth. If the dentist can open through the root canal well enough and treat it, thus getting rid of the infection, and then fill it properly, the tooth would not have to be extracted, but if there is any amount of caries which cannot be gotten rid of, extraction is necessary.

Dr. Fletcher asked if the very modern dentists do not actually go through the soft parts into the bone. Dr. Carmody replied that dentists do this very frequently, and cut off the end of the root. Very frequently he makes a fistulous opening. In that way he simply drains the cavity from both ways, and in the case of a lower tooth it is very convenient to do this, because you can get better drainage then through the tooth.

As regards dermoid cysts, the probabilities are that a dentigerous cyst is very similar to a dermoid cyst; however, it is not a change like we have in dermoids. In the dermoid, of course, we have the development of hair and teeth, while in the case of dentigerous cyst we simply have, as a rule, a part from one of the other teeth, or a normal tooth which has gotten into the maxillary sinus, or into a region where it cannot erupt itself, and is forced in there. The irritation probably has something to do with the production of the cystic fluid.

Dr. Robertson spoke about leukoplakia, and Dr. Carmody believed that if he would look up all the literature on the subject, he would find that smoking has something to do with it, nevertheless. There is no question but what teeth have a great deal to do with epithelioma in the mouth. Rough teeth very frequently produce epithelioma without leukoplakia at all. He has seen one case of socalled leukoplakia of the lips where there were no teeth present, and had not been for a number of years. The leukoplakia occurred after the removal of the teeth. That was not said in denial of Dr. Robertson's statement, but that is not the opinion of most people. Dr. Carmody believes, however, that most of the cases of epithelioma of the mouth are due to either bad plates, bad bridges, broken-down teeth, or something of that kind, and the smoking is just another irritant. Frequently epithelioma of the lip is not due only to smoking, but to a displacement of some cells in shaving at times, because of cutting of the lips, etc., that probably forces in some epithelium into the lower stratum of the lip. That, however, was only his own idea, and he did not know whether it was really so or not.

Dr. Kahn spoke of broadening of the arches. That was simply mentioned by him in the paper, because it had been covered in so many cases. Nevertheless, he believes that a great many septa can be straightened in this way, and the little patients saved a great deal of trouble.

Another thing to be remembered is that the lymphatics in younger patients are open more than in older patients. The speaker had the misfortune to have a patient die of meningitis following a submucous resection, and since that time he has hesitated to operate on young children. This patient was only eleven years of age. Someone asked how late these operations could be performed. The oldest was twenty-eight years. The probabilities are that spreading the arch and helping to straighten the septum, whether by drawing it down forcibly or simple stimulation of the growth of the bone of the palate, meets with better success if done earlier. Orthodontists believe now in beginning with the temporary set of teeth, if there seems to be trouble, and especially after the eruption of the six-year molars. Those are erupted very often before the fifth year, and regulation of the teeth can be begun at that time, so as to straighten the teeth, and, of course, it will help the nose a great deal more than if done later.

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XLV.

CLINICAL PATHOLOGY OF TUBERCULOUS LARYNGITIS.

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In 1914 the writer[1] presented a paper on the Etiology and Prophylaxis of Tuberculous Laryngitis. It was then stated that an intensive study of laryngeal tuberculosis presented so many important aspects that its results could not possibly be detailed in one paper. It appeared to the writer that the subject could more profitably be discussed by dividing it into sections, and this communication will therefore be devoted to the study of the clinical pathology of laryngeal tuberculosis.

It can readily be seen that a thorough knowledge of clinical pathology is fundamental. Without a full conception of the various phases and course of a lesion, we are scientifically unable to prognosticate and treat a disease.

The prognosis of laryngeal tuberculosis, although not easily determined, depends on many influencing factors: the pulmonary lesion, general condition of the patient, site and extent of the laryngeal lesion, early diagnosis, character of treatment, etc. To the author, however, it seems that one of the major influencing factors is the character of the lesion itself. Dr. Robert Levy[2] of Denver says: "The advent of laryngeal complication adds to the gravity of the prognosis. This statement applies only to those cases in which the nature of the complication is of the rapidly active type with marked edematous swelling and abundant tubercles." He further states that "in that form of laryngeal tuberculosis in which there is little or no tendency to ulceration, the form characterized by more or less firm infiltration, the progress of the general disease is not materially affected."

In the above quotation regarding the prognosis, the weight attached to the character of the lesion makes a strong impression; its importance could not possibly be exaggerated and is in accord with the author's views. In pulmonary tuberculosis one is immediately impressed by the difference in gravity between a case of acute tuberculosis and fibroid phthisis, because of the difference in pathology. Similarly in tuberculosis of the larynx, in addition to a consideration of all other factors, an attempt should be made at arriving at a prognosis by taking into account the pathology.

The author's experience has taught him to classify laryngeal tuberculosis as acute, subacute, and chronic.

THE ACUTE TYPE (Figures 7 and 12), the least frequent of the series, is characterized by a soft edema of the larynx with a marked tendency to ulceration. It may begin acutely or else follow the subacute or chronic type. It usually occurs in advanced pulmonary cases, often in moderately advanced, and occasionally in the incipient cases. There is little or no tendency to fibrosis, and it therefore takes an acute course. Nodules, consisting of tubercles, can often be seen projecting above the surface of the mucosa. When the tubercles caseate, they give rise to ulcers. During the stage of caseation the nodules often coalesce with the production of extensive ulcerations. The patient usually suffers from marked hoarseness or aphonia, sensation of fullness in throat and irritation with constant desire to cough and expectorate. Dysphagia is of common occurrence, and may be very pronounced, depending largely on the location and character of the ulcerations. With the patient in good general condition it may run its course, become subacute and occasionally arrested, but in general the lesion is hardly amenable to treatment. As to prognosis, the life of the patient is generally from six to eighteen months. It is often a terminal condition in advanced pulmonary cases, especially in those that are overwhelmed by the toxins of the tubercle, and that have completely lost their acquired immunity to the disease. This is probably the type which the older writers had in mind when speaking of laryngeal tuberculosis as the "death warrant."

THE SUBACUTE TYPE (Figures 1, 2, 3, 4, 5, 6, 9, 10 and 13) is characterized by a pseudoedema of part or parts of the larynx with a moderate tendency to fibrosis. It may begin as such or else follow the acute or chronic type. It takes a subacute course, has a tendency to fibrosis, and is usually benefited by treatment. Papilliform infiltrates and soft polypoid excrescences usually belong to this type. The local symptoms may be slight or moderate, depending largely on the extent and site of the lesion. Hoarseness as a rule is present, particularly when the true vocal cords or the interarytenoid sulcus is involved. Dryness in the larynx is usually complained of, especially when the fibrosis is extensive. With the patient in good general condition it runs its course and may become completely arrested. The prognosis as to life and voice depends much on the patient's general condition and on an early diagnosis. With proper treatment the prognosis in the average subacute case is favorable.

THE CHRONIC TYPE (Figures 8 and 11), which offers the best outlook, is characterized by firm infiltration of part or parts of the larynx, with a marked tendency to fibrosis and healing. The lesion, due to an excessive proliferation of connective tissue cells, is usually limited in extent in the affected areas, and may remain so for an indefinite period. These patients suffer very little, and often, with the exception of occasional dryness in the throat and slight hoarseness, have no symptoms at all. For this reason this type is very often overlooked. The prognosis as to life and voice is very good, and unless it takes on a course of the acute or subacute type, will have very little influence on the pulmonary condition. The diagnosis of this type, however, is of major importance. The patient should be told of the condition, so that the proper measures may be taken. Abuse of the larynx will often modify its course, while with proper attention this condition will usually remain secondary to the pulmonary lesion.

The three above described types, although one may follow or precede the other, are undoubtedly distinct, and it is highly essential to differentiate them on account of the bearing on prognosis and treatment. As a rule, more than one examination will be required to make the proper diagnosis and to determine the course of the disease.

The author has therefore observed and studied one hundred cases of laryngeal tuberculosis over a period of three months to two years, and has carefully noted the affected parts in detail. Only those cases that presented a laryngeal lesion without doubt, and only those that offered an opportunity for a complete examination, were included in this study. Otherwise the cases were not especially selected.

All cases were examined by indirect laryngoscopy. Where the character of the lesion was still in doubt after the examination with the ordinary mirror, a magnifying laryngoscope was used, thus eliminating possible errors.

The result of this study will now be given in detail.

SEX.

Sixty-four of the patients were males, while thirty-six were females. These figures do not express the comparative frequency of tuberculous laryngitis in the sexes. In past work the author found tuberculous laryngitis one and a half times as common in males as in females.

AGE.

Fourteen of the patients were between fifteen and twenty years of age, only one of these being fifteen, and the rest over eighteen. Fifty-three of the patients were between twenty-one and thirty. Twenty-one were between thirty-one and forty, while twelve were between forty-one and fifty. The majority of the patients, therefore, were between twenty and thirty years of age.

SPUTUM.

The sputum contained tubercle bacilli in all cases.

PULMONARY STAGE.*

Twelve of the patients were in the incipient stage, fifty-nine in the moderately advanced, and twenty-nine in the far advanced. As only about fifteen per cent of our cases are in the advanced stage, the reason for the apparently lesser frequency of laryngeal tuberculosis in the advanced cases is obvious.

LARYNGEAL STAGE.†

Sixty cases showed early involvement of the larynx, and were classified as stage one; thirty-three cases showed moderate involvement, and were classed as stage two; while seven cases had an advanced involvement of the larynx, and were put into stage three. The high percentage of early involvement shows the importance of examination of the larynx in all cases of pulmonary tuberculosis. If the larynx is examined only when the patient complains of marked symptoms, the probabilities are that the lesion has already passed its incipiency and is in an advanced stage. Symptoms are usually absent or very slight during the early stage of infiltration, and only by thorough search may early lesions be detected.

CLINICAL TYPES.

Nine of the cases were of the acute type, fifty-two of the subacute, and thirty-nine of the chronic. The low percentage of the acute type is partly due to the fact that only seven or eight per cent of all the cases in the sanatorium are bed patients.

EPIGLOTTIS.

The epiglottis was found clinically normal in sixteen of the cases, marked congestion was observed in forty-two, and although considered pathologic, was classified as such but not as clinically tuberculous. The author has no doubt that if microscopic sections were taken, at least some of these would be found tuberculous, although clinically they appeared to be catarrhal. Anemia of the epiglottis was present in fourteen cases, and here the author wishes to call attention to the apparent infrequency of anemia as compared with congestion. In most of these cases the anemia was only a local manifestation of the general anemia and not of any pathologic process per se. Slight infiltration was detected in twenty cases. Marked infiltration in three cases, while marked infiltration with single or multiple erosions was present in five cases. The site of the involvement was either at the rim of the epiglottis or else at the cushion. All the lesions were bilateral and more or less diffuse. Only one case presented an infiltration limited to the right side and to the lingual surface of the epiglottis. (Figure 6.) In two cases, with the exception of slight interarytenoid hyperplasia, the infiltration was limited to the epiglottis. Thus it is seen that clinically, of the one hundred cases examined, twenty-eight showed an involvement of the epiglottis.

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*Pulmonary stages according to the Classification of the National Association for the Study and Prevention of Tuberculosis.

†Laryngeal lesions classified according to author's classification. Annals of Otology, Rhinology and Laryngology, December, 1914.

INTERARYTENOID SPACE.

The interarytenoid sulcus was found clinically negative in five cases. Four cases showed a congestion. Slight infiltration was observed in forty-one cases, while twenty-five cases exhibited marked infiltration. Infiltration with erosion was observed in eighteen of the cases. Papillomatous growths were noticed in six cases, while one case showed a tuberculoma. (Figure 8.) The patient with the tuberculoma gives a history of about five years' duration. He has been observed by the author for the past two years with apparently no change in lesion. From the above, then, we learn that of the one hundred cases studied, ninety-one showed more or less involvement of the interarytenoid sulcus. This frequency of occurrence is easily explained by the fact that the interarytenoid space is a vulnerable point, due to its location and anatomic arrangement, making it subject to more traumatism than any other part of the larynx. The infiltration was generally of the broad base type, and usually confined to the middle of the sulcus. The color varied from a grayish tint to the redness of congestion. In a large number mucopurulent secretion could be seen adhering to the space. The "median fissure" was frequently observed, while the "lateral furrow," as described by Casselberry,[3] was quite common. The infiltration was limited to the space in six cases.

ARYEPIGLOTTIDEAN FOLDS.

The aryepiglottidean folds were found clinically negative in sixty-two cases. Eighteen of the cases showed a bilateral congestion. Bilateral infiltration was present in seventeen cases. One case presented unilateral infiltration of the right fold, while in another the infiltration was limited to the left fold. In one case of the acute type both folds were markedly infiltrated and eroded. Thus we learn that infiltration of the folds was present in twenty cases. Of the twenty affected cases, eleven were in conjunction with a bilateral infiltration of the arytenoids. Four were associated with an infiltration of the epiglottis and arytenoids. One in conjunction with epiglottidean involvement. While the remaining four occurred together with involvement of one or more of the following structures: the interarytenoid space, ventricular bands, and vocal cords or processes. The above would seem to suggest that the folds are affected mainly by extension from the arytenoids or the epiglottis, and that the infiltration very seldom originates there.

ARYTENOID CARTILAGES.

The arytenoids were found clinically normal in thirty-six cases; eight of the cases showed a bilateral congestion. Bilateral infiltration was noted in forty of the cases. Unilateral infiltration was observed in twelve instances—eight of the right arytenoid and four of the left. Marked bilateral infiltration with erosion was noticed in three, while unilateral infiltration with erosion was seen in one. We then note that fifty-six of the series showed more or less infiltration of one or both arytenoid cartilages. Of these, forty-three had bilateral involvement, while in thirteen only was the infiltration limited to one side. Its high frequency of occurrence can partly be attributed to the prominent part played by the arytenoids in all expiratory acts, as in phonation, coughing, sneezing, etc.

VENTRICULAR BANDS.

The ventricular bands were found clinically normal in sixty-six cases. Seven cases showed a bilateral congestion. In fourteen of the cases a bilateral infiltration was observed, while unilateral infiltration was seen in twelve cases—four on the right and eight on the left. One case presented a marked bilateral infiltration with erosion. Thus twenty-seven cases showed affection of the ventricular bands. It was also noted that the infiltration of the bands was generally diffuse.

TRUE VOCAL CORDS.

The true vocal cords were found clinically negative in thirty-four cases. Twenty of the cases presented a bilateral congestion, while a unilateral congestion was noticed in four cases—two of the left cord and two of the right. Slight bilateral infiltration was observed in eleven cases. Slight unilateral infiltration in three, all confined to the right vocal cord. Marked bilateral infiltration was observed in seven cases, while marked unilateral infiltration was seen in two—one of the right and one of the left. Bilateral infiltration with erosion was observed in twelve cases, and unilateral infiltration with erosion was seen in seven cases, four of which were of the right cord and three of the left. The above shows that forty-two out of the one hundred cases studied showed involvement of the true vocal cords. Of these, thirty were bilateral while twelve were unilateral.

VOCAL PROCESSES.

The vocal processes were found to be affected early in the disease, and therefore were studied separately. Thirty-one of the cases presented a clinically normal appearance, while nine showed a bilateral congestion. Bilateral infiltration was observed in twenty-seven cases, while nine cases showed unilateral involvement—seven of the left and two of the right vocal process. Bilateral infiltration with erosion was observed in seventeen cases, while seven showed unilateral infiltration with erosion, of which five were of the right and two of the left. Thus we see that sixty cases out of one hundred showed involvement of the vocal processes, of which forty-four were bilateral and sixteen unilateral. The above indicates that the vocal processes were affected about as frequently as the arytenoids. It is of interest to note that affection of the vocal processes was associated with involvement of the other parts of the larynx, as follows: with the true cords in nineteen cases; with the arytenoids in eleven; with arytenoids and cords in twenty-one; and with the interarytenoid sulcus in nine. The proximity of the vocal processes to the arytenoids, true cords and interarytenoid space with the consequent trauma it is subjected to would readily explain the high frequency of involvement.

The foregoing discussion may be summarized as follows:

1. A clear conception of the nature of a lesion is of utmost importance. Without it we are unable scientifically to prognosticate and treat a disease.
2. The character of the lesion is one of the chief determining factors in prognosis.
3. In tuberculosis of the larynx the pathology does not differ in any way from the pathology of tuberculosis of the lungs.
4. There are three distinct types of laryngeal tuberculosis: acute, subacute, and chronic.
5. Cases of the acute and subacute type have symptoms referable to the larynx, and the diagnosis, as a rule, is easily made. On the other hand, cases of the chronic type very seldom suffer from annoying symptoms, and only on close scrutiny can the lesion be detected.
6. One of the important conclusions arrived at is that the frequency with which a part is affected is in direct proportion to the amount of trauma that part receives as a result of its location and its fuctional activity. The above is fully corroborated by the results of the study presented, in which it is shown that the most frequent site of involvement is the interarytenoid space, followed in order of frequency by the vocal processes, arytenoid cartilages, vocal cords, ventricular bands, aryepiglottidean folds, and epiglottis.

TABLE SHOWING IN DETAIL THE RESULTS OF THE STUDY OF ONE HUNDRED CASES OF LARYNGEAL TUBERCULOSIS.

SEX.

Males	64
Females	36
Total	100

AGE.

Between 15 and 20	14	
Between 21 and 30	53	
Between 31 and 40	21	
Between 41 and 50	12	
Total		100

SPUTUM.

Positive	100

PULMONARY STAGE.*

Incipient	12	
Moderately advanced	59	
Far advanced	29	
Total		100

LARYNGEAL STAGE.†

First stage	60	
Second stage	33	
Third stage	7	
Total		100

CLINICAL TYPES.

Acute type	9	
Subacute	52	
Chronic	39	
Total		100

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*See Classification of the National Association for the Study and Prevention of Tuberculosis.

†See author's Classification of Laryngeal Tuberculosis.

EPIGLOTTIS.

Negative	16	
Congested	42	
Anemic	14	
Clinically nontuberculous total		72
Slight infiltration	20	
Marked infiltration	3	
Infiltration with erosion	5	
Tuberculous total		28
		100

INTERARYTENOID SPACE.

Negative	5	
Congestion	4	
Clinically nontuberculous total		9
Slight infiltration	41	
Marked infiltration	25	
Infiltration with erosion	18	
Papillomatous growths	6	
Tuberculoma	1	
Tuberculous total		91
		100

Note.—Infiltration limited to interarytenoid space in six cases.

ARYEPIGLOTTIDEAN FOLDS.

Negative	62	
Congested	18	
Clinically nontuberculous total		80

Slight infiltration	17	
Infiltration right	1	
Infiltration left	1	
Infiltration with erosion	1	
Tuberculous total		20
		100

Note.—Associated lesions:

With infiltration of arytenoids	11
With epiglottis and arytenoids	4
With epiglottis	1
With interarytenoid space, ventricular bands, vocal cords or processes	4

ARYTENOID CARTILAGES.

Negative	36	
Congested	8	
Clinically nontuberculous total		44
Infiltration right and left	40	
Infiltration right	8	
Infiltration left	4	
Infiltration with erosion, right	3	
Infiltration with erosion, left	1	
Tuberculous total		56
		100

Note.—

Bilateral involvement	43
Unilateral involvement	13

VENTRICULAR BANDS.

Negative	66	
Congested	7	
Clinically nontuberculous total		73

Infiltration right and left	14	
Infiltration right	4	
Infiltration left	8	
Infiltration with erosion	1	
Tuberculous total		27
		100

Note.—

Bilateral involvement	15
Unilateral involvement	12

VOCAL CORDS.

Negative	34	
Congestion	20	
Congestion right	2	
Congestion left	2	
Clinically nontuberculous total		58
Slight infiltration	11	
Slight infiltration right	3	
Marked infiltration	7	
Marked infiltration right	1	
Marked infiltration left	1	
Infiltration with erosion	12	
Infiltration with erosion, right	4	
Infiltration with erosion, left	3	
Tuberculous total		42
		100

Note.—

Bilateral involvement	30
Unilateral involvement	12

VOCAL PROCESS.

Negative	31	
Congested	9	
Clinically nontuberculous total		40

Infiltration right and left	27	
Infiltration right	2	
Infiltration left	7	
Infiltration with erosion	17	
Infiltration with erosion, right	5	
Infiltration with erosion, left	2	
Tuberculous total		60
		100

Note.—	
Bilateral involvement	44
Unilateral involvement	16
Associated lesions:	
With true vocal cords	19
With arytenoid cartilages	11
With arytenoids and cords	21
With interarytenoid sulcus	9

TABLE SHOWING ORDER OF FREQUENCY OF INVOLVEMENT OF THE DIFFERENT PARTS OF THE LARYNX.

	Tuberculous.	Clinically Nontuberculous.
Interarytenoid space	91	9
Vocal processes	60	40
Arytenoid cartilages	56	44
Vocal cords	42	58
Epiglottis	28	72
Ventricular bands	27	73
Aryepiglottidean folds	20	80

AUTHOR'S CLASSIFICATION OF TUBERCULOUS LARYNGITIS.

Class 1.—Stage of infiltration.

1. Positive suptum. Negative in exceptional cases.
2. Recurrent or persistent hoarseness.
3. Dysphagia present or absent.
4. Signs of infiltration of any or all the parts of the larynx, especially interarytenoid space, vocal cords, aryepiglottidean folds, ventricular bands, arytenoid cartilages, or epiglottis.

Class 2.—Stage of edema with superficial erosion.

1. Positive sputum.
2. Persistent hoarseness.
3. Dysphagia present or absent.
4. Dysphonia present or absent.
5. Signs of tumefaction of part of the larynx with superficial erosion.

Class 3.—Advanced stage.

1. Positive sputum.
2. Marked hoarseness or aphonia.
3. Dysphagia present or absent.
4. Dysphonia present or absent.
5. Marked tumefaction and ulceration of any or parts of the larynx.
6. Perichondritis sometimes present.*

AUTHOR'S CLASSIFICATION OF TUBERCULOUS LARYNGYTITIS ON DISCHARGE.

Apparently Cured.—All laryngeal symptoms absent for a period of two years under ordinary conditions of life; the physical signs to be those of a healed lesion.

Arrested.—All laryngeal symptoms absent for a period of six months; the physical signs to be those of a healed lesion.

Apparently Arrested.—All laryngeal symptoms absent for a period of three months; the physical signs to be those of a healed lesion.

Quiescent.—All laryngeal symptoms absent for a period of two months; physical signs stationary or retrogressive.

Improved.—Laryngeal symptoms lessened or entirely absent; physical signs improved or unchanged.

Unimproved.—All essential symptoms and signs unabated or increased.

CLASSIFICATION OF PULMONARY TUBERCULOSIS ACCORDING TO THE NATIONAL ASSOCIATION FOR THE STUDY AND PREVENTION OF TUBERCULOSIS.

INCIPIENT.

Slight or no constitutional symptoms (including particularly gastric or intestinal disturbances or rapid loss of weight).

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*Hoarseness, dysphonia and aphonia are usually absent in tuberculosis involvement limited to the epiglottis.

Slight or no elevation of temperature or acceleration of pulse at any time during the twenty-four hours.

Expectoration usually small in amount or absent.

Tubercle bacilli may be present or absent.

Slight infiltration limited to the apex of one or both lungs, or a small part of one lobe.

No tuberculous complications.

MODERATELY ADVANCED.

No marked impairment of function, either local or constitutional.

Marked infiltration more extensive than under incipient, with little or no evidence of cavity formation.

No serious tuberculous complications.

FAR ADVANCED.

Marked impairment of function, local and constitutional.

Extensive localized infiltration or consolidation in one or more lobes.

Or disseminated areas of cavity formation.

Or serious tuberculous complications.

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No.	Name	Age	Sputum	Pulm. Stage	Laryn. Stage	Type	Epiglottis	Aryepiglottic Folds	Arytenoid Cartilage	Interarytenoid Space	Ventricular Bands	Vocal Cords	Vocal Process
1	T. C.	24F	Positive	3	2	Subacute	Congested	Infiltration	Infiltration	Marked infiltration	Infiltration	Congested	Negative
2	P. H.	22M	"	2	2	"	"	"	"	Infiltration, erosion	Infiltration L.	Infiltration, erosion	Infiltration
3	S. G.	21M	"	2	1	"	"	Infiltration L.	Infiltration L.	Slight infiltration	Negative	Negative	Negative
4	T. H.	23M	"	3	1	"	"	Congested	Infiltration R.	Congested	Infiltration L.	"	"
5	S. L.	24F	"	3	3	"	Anemic	Infiltration	Negative	Papillomata	Infiltration	Marked infiltration	Infiltration
6	F. L.	42F	"	2	2	"	"	"	"	Marked infiltration	Infiltration R.	Infiltration, erosion	Infiltration, erosion L.
7	C. B.	20F	"	2	3	Acute	Slight infiltration	"	Infiltration, erosion	Infiltration, erosion	Negative	"	Infiltration, erosion
8	G. A.	37F	"	2	1	Subacute	"	Negative	Infiltration	Marked infiltration	"	Slight infiltration	Infiltration
9	B. T.	30F	"	2	2	"	Congested	Congested	Negative	Papillomata	"	Infiltration, erosion R.	Infiltration, erosion R.
10	McM.	18F	"	2	2	"	Anemic	Negative	Infiltration	Infiltration, erosion	"	Marked infiltration	Infiltration
11	B. I.	25F	"	3	1	Chronic	Congested	"	Negative	Slight infiltration	"	Congested	Negative
12	C. G.	22F	"	2	3	Acute	Marked infiltration	Infiltration	Infiltration, erosion L.	Marked infiltration	"	Infiltration, erosion L.	Infiltration
13	K. S.	18F	"	3	1	Chronic	Negative	Negative	Negative	"	"	Infiltration, erosion	Infiltration, erosion
14	S. R.	27F	"	2	1	"	"	"	Infiltration	Slight infiltration	"	Congested	Infiltration L.
15	T. C.	18F	"	2	1	"	Anemic	"	"	"	"	Negative	Infiltration

No.	Name	Age	Sputum	Pulm. Stage	Laryn. Stage	Type	Epiglottis	Aryepiglottic Folds	Arytenoid Cartilage	Interarytenoid Space	Ventricular Bands	Vocal Cords	Vocal Process
16	S. M.	20F	Positive	3	1	Subacute	Congested	Negative	Congested	Marked infiltration	Negative	Negative	Negative
17	D. J.	27F	"	2	1	Chronic	Anemic	"	"	Slight infiltration	"	Congested	"
18	McL.R	30F	"	3	1	Subacute	"	Infiltration	Infiltration	"	Infiltration R.	Negative	"
19	E. J.	27M	"	1	2	"	Infiltration, erosion	Negative	"	Negative	Negative	Slight infiltration	"
20	V. W.	32M	"	2	1	"	Anemic	"	"	"	"	"	Infiltration
21	C.	24M	"	2	3	"	"	"	"	Marked infiltration	Infiltration	Infiltration, erosion	Infiltration, erosion
22	B. G.	25M	"	2	2	"	Slight infiltration	"	Negative	Infiltration, erosion	Negative	"	"
23	K. J.	46M	"	2	2	"	Congested	"	"	Slight infiltration	"	"	"
24	R. H.	24M	"	3	2	"	Anemic	Infiltration	Infiltration	Marked infiltration	"	Infiltration, erosion R.	Infiltration, erosion R.
25	W. M.	19F	"	1	1	Chronic	"	Negative	Negative	Negative	"	Slight infiltration	Slight infiltration
26	Q. M.	22F	"	3	1	Subacute	Congested	"	Slight infiltration	Slight infiltration	"	Negative	Infiltration
27	M. C.	23F	"	2	1	Chronic	"	"	Negative	Slight infiltration	"	Congested	"
28	M. M.	21M	"	1	2	Acute	Slight infiltration	"	"	Infiltration, erosion	Infiltration	Marked infiltration	"

29	S. J.	42M	Positive	3	1	Chronic	Congested	Negative	Negative	Negative	Infiltration L.	Negative	Negative
30	F. J.	28M	"	3	1	"	Slight infiltration	"	Infiltration	Slight infiltration	Negative	"	Congested
31	W. L.	22M	"	3	2	Subacute	Congested	"	Infiltration R	Marked hyperplasia	"	"	Infiltration, erosion
32	G. J.	27M	"	2	1	"	"	Infiltration	Infiltration	"	"	"	Infiltration
33	M. C.	24M	"	2	1	"	"	Negative	"	Congested	"	Slight infiltration	Infiltration L.
34	B. K.	23F	"	3	1	Chronic	Anemic	"	Infiltration R	Slight infiltration	"	Slight infiltration R.	Negative
35	K. B.	27F	"	2	2	Subacute	Slight infiltration	"	"	Papillomata	"	Negative	"
36	R. J.	20F	"	2	1	Chronic	Congested	Congested	"	Slight infiltration	Congested	"	"
37	F. P.	18M	"	2	1	Subacute	Anemic	Negative	"	Slight hyperplasia	Negative	"	"
38	R. B.	23M	"	3	1	"	"	"	Infiltration L	Marked hyperplasia	"	Marked infiltration L.	Infiltration L.
39	T. S.	29M	"	2	2	"	Congested	Infiltration	Infiltration	Infiltration, erosion	"	Negative	Negative
40	K. H.	44M	"	2	1	"	"	Congested	Congested	Congested	"	Slight infiltration R.	"
41	M. B.	31F	"	2	1	Chronic	"	Negative	Negative	Slight infiltration	"	Negative	"
42	C. L.	27F	"	2	1	Subacute	"	Congested	Congested	"	"	Congested L.	Congested

No.	Name	Age	Sputum	Pulm. Stage	Laryn. Stage	Type	Epiglottis	Aryepiglottic Folds	Arytenoid Cartilage	Interarytenoid Space	Ventricular Bands	Vocal Cords	Vocal Process
43	B. L.	31F	Positive	2	1	Subacute	Congested	Infiltration R	Infiltration	Slight hyperplasia	Negative	Congested L.	Negative
44	C. J.	39M	"	2	3	Chronic	Negative	Negative	Negative	Tuberculoma	Infiltration R.	Marked infiltration	Infiltration
45	D. J.	49M	"	2	2	"	Infiltration, erosion	"	"	Marked hyperplasia	Negative	Negative	Negative
46	G. J.	45M	"	3	1	"	Congested	Congested	Infiltration	Slight hyperplasia	"	Congested R.	Congested
47	C. J.	29M	"	1	1	Subacute	Slight infiltration	Negative	"	Congested	Congested	Congested	"
48	K. I.	44M	"	2	1	Chronic	Slight infiltration	"	Slight infiltration	Slight infiltration	"	Negative	Negative
49	C. F.	28M	"	2	1	"	Congested	"	Infiltration R.	"	Infiltration L.	Congested	Infiltration L.
50	P. A.	34F	"	2	1	"	Anemic	"	Negative	Infiltration, erosion	Infiltration	Negative	Negative
51	M. B.	23F	"	2	1	"	Negative	"	"	Slight infiltration	Negative	"	"
52	D. M.	32F	"	2	1	"	"	"	Infiltration	"	"	"	"
53	S. E.	19F	"	2	2	Subacute	Slight infiltration	"	"	"	Infiltration	Infiltration, erosion L.	Infiltration, erosion
54	R. S.	33F	"	3	1	Chronic	Negative	"	Slight infiltration	"	Negative	Negative	Infiltration L.
55	B. C.	22F	"	2	1	Subacute	Slight infiltration	"	Negative	Marked infiltration	"	Slight infiltration	Infiltration

56	P. J.	29M	Positive	2	1	Subacute	Congested	Infiltration	Negative	Papillomata	Infiltration L.	Negative	Negative
57	B. W.	47M	"	2	2	"	Negative	Negative	"	Marked infiltration	Negative	Infiltration, erosion R.	Infiltration, erosion R.
58	W. A.	50F	"	2	2	"	"	"	"	Slight infiltration	"	Negative	Infiltration, erosion
59	B. G.	30M	"	2	1	Chronic	Congested	"	"	"	Slight infiltration	"	Infiltration
60	G. J.	28M	"	2	1	Subacute	"	"	Infiltration	"	Negative	"	Negative
61	A. J.	24M	"	2	2	"	"	"	Negative	Marked infiltration	"	Infiltration, erosion	Infiltration, erosion
62	M. M.	21M	"	2	1	Chronic	Slight infiltration	"	Infiltration	"	"	Congested	Congested
63	L. F.	45M	"	3	1	"	"	Congested	"	Slight infiltration	Congested	"	"
64	L. W.	22M	"	1	1	"	Negative	Negative	Negative	"	Negative	Negative	Infiltration
65	K. S.	24M	"	1	1	"	Congested	Congested	Infiltration	"	Infiltration	"	Negative
66	W. D.	19M	"	3	1	Subacute	"	"	Congested	"	Infiltration R.	"	"
67	S. E.	20M	"	1	1	Chonic	"	"	Infiltration	"	Negative	Congested	Congested
68	P. R.	31M	"	3	3	Acute	Infiltration, erosion	Infiltration, erosion	Infiltration, erosion	Infiltration, erosion	Infiltration	Infiltration, erosion	Infiltration, erosion

No.	Name	Age	Sputum	Pulm. Stage	Laryn. Stage	Type	Epiglottis	Aryepiglottic Folds	Arytenoid Cartilage	Interarytenoid Space	Ventricular Bands	Vocal Cords	Vocal Process
69	R. W.	35M	Positive	2	1	Chronic	Congested	Negative	Negative	Negative	Negative	Infiltration R.	Infiltration R.
70	R. L.	22M	"	2	2	Subacute	Slight infiltration	"	"	Infiltration, erosion	"	Infiltration, erosion R.	Infiltration, erosion R.
71	R. M.	22M	"	1	1	Chronic	Congested	"	"	Marked infiltration	"	Slight infiltration	Infiltration
72	F. J.	35M	"	3	1	Subacute	Slight infiltration	"	"	"	Infiltration	Congested	"
73	W. C.	40M	"	3	1	"	Congested	Congested	Infiltration	"	"	Slight infiltration	"
74	H. C.	42M	"	2	2	"	Slight infiltration	Negative	Negative	Infiltration, erosion	Negative	"	"
75	K. B.	21M	"	1	1	"	"	Congested	Infiltration	Slight infiltration	Congested	Congested	"
76	R. P.	45M	"	3	1	Chronic	Negative	Negative	"	"	Infiltration L.	Negative	Negative
77	C. J.	33M	"	1	2	Subacute	"	"	Infiltration L.	Papillomata	Negative	Slight infiltration	Infiltration
78	W. E.	22F	"	2	1	Chronic	Slight infiltration	Slight infiltration	Negative	Slight infiltration	"	Negative	Infiltration L.
79	M. A.	27M	"	3	2	Subacute	Marked infil. erosion R	Negative	"	"	"	"	Negative
80	M. B.	19F	"	2	2	Acute	Slight infiltation	"	Infiltration	Infiltration, erosion	"	Infiltration, erosion	Infiltration, erosion

81	Y. E.	32M	Positive	2	3	Acute	Marked infiltra., erosion	Infiltration	Marked infiltra., erosion	Infiltration, erosion	Infiltration, erosion	Infiltration, erosion	Infiltration, erosion
82	D. E.	39M	"	3	2	Subacute	Negative	Negative	Negative	Marked infiltartion	Negative	"	Infiltration
83	H. I.	37M	"	2	1	Chronic	"	"	"	Infiltration, erosion	"	Marked infiltration R.	Infiltration, erosion R.
84	G. J.	21M	"	2	1	Subacute	Congested	"	Infiltration	Slight infiltration	"	Congested R.	Negative
85	S. S.	26M	"	1	1	Chronic	Negative	"	Negative	"	"	Slight infiltration R.	Infiltration, erosion L.
86	L. J.	23M	"	3	2	Subacute	"	"	Infiltration L.	Papillomata	"	Negative	Infiltration
87	G. H.	25M	"	3	1	Chronic	Slight infiltration	"	Negative	Slight infiltration	"	Congested	Negative
88	S. P.	21M	"	3	2	Acute	Marked infiltration	Infiltration	Infiltration	Infiltration, erosion	"	"	Infiltration
89	F. W.	34M	"	3	2	"	Congested	"	"	"	Congested	Infiltration, erosion	Infiltration, erosion
90	C. C.	28M	"	2	2	Subacute	"	Congested	"	"	Infiltration	Congested	"
91	K. A.	38F	"	2	2	"	Marked infiltration	Infiltraton	"	Marked infiltration	Negative	Slight infiltration	"
92	M. S.	18F	"	2	1	Chronic	Congested	Negative	Negative	Slight infiltration	"	Negative	Infiltration L.
93	M. L	30F	"	2	2	Acute	"	Infiltration	Congested	Marked infiltration	Infiltration	Marked infiltration	Marked infiltration

No.	Name	Age	Sputum	Pulm. Stage	Laryn. Stage	Type	Epiglottis	Aryepiglottic Folds	Arytenoid Cartilage	Interarytenoid Space	Ventricular Bands	Vocal Cords	Vocal Process
94	R. L.	34M	Positive	2	1	Chronic	Congested	Congested	Congested	Marked infiltration	Negative	Negative	Negative
95	R. G.	21M	"	3	1	"	Negative	Negative	Infiltration R	Slight infiltration	"	Congested	"
96	H. J.	25M	"	2	1	Subacute	Congested	Congested	Infiltration	"	Infiltration L.	"	Infiltration R.
97	R. L.	15M	"	1	1	Chronic	"	Negative	Negative	Marked infiltration	Infiltration	"	Congested
98	E. A.	21M	"	2	2	"	Slight infiltration	Congested	Congested	Infiltration, erosion	Congested	"	"
99	B. W.	34M	"	2	2	Subacute	Congested	"	Infiltration	Marked infiltration	Negative	Marked infiltration	Infiltration, erosion
100	G. F.	35M	"	2	2	"	"	"	"	Infiltration, erosion	Infiltration L.	Congested	"

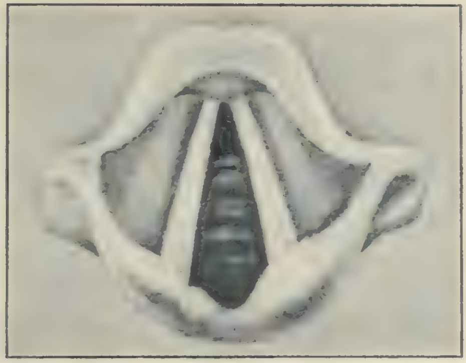

FIGURE 1.

S. G., aged twenty-one years. Sputum, positive. Pulmonary stage, second. Infiltration limited to left arytenoid and cartilage of Wrisberg. Slight superficial erosion can be seen Patient in good general condition, no hoarseness. Occasional dryness and tickling in larynx. Approximation of cords good. Diagnosis: Subacute tuberculosis of the larynx.

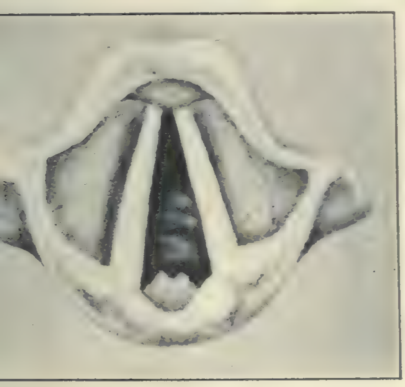

FIGURE 2.

aged thirty-two years. Sputum, posi-
ulmonary stage, first. Papillomatous in-
n of posterior commissure. Slight thick-
f left arytenoid and cartilage of Wris-
Both cords slightly infiltrated and
l. Constant dryness and irritation in
Marked hoarseness for past three
Wasserman four plus. Approximation
s incomplete. Diagnosis: Subacute tu-
sis of the larynx.

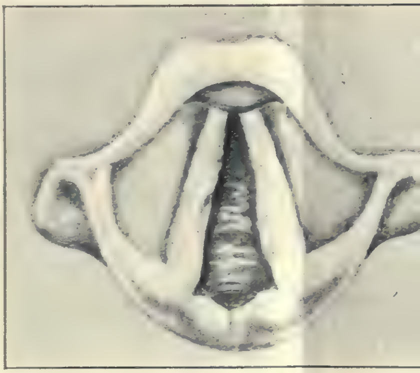

FIGURE 3.

K. J., aged forty-five years. Sputum, p
Pulmonary stage, second. Marked infil
of both vocal cords and both vocal pro
Cleft-like ulceration of right vocal cord.
interarytenoid thickening with medium
Slight infiltration of left arytenoid. Appr
tion incomplete on phonation. Marked
ness. Diagnosis: Subacute tuberculo
the larynx.

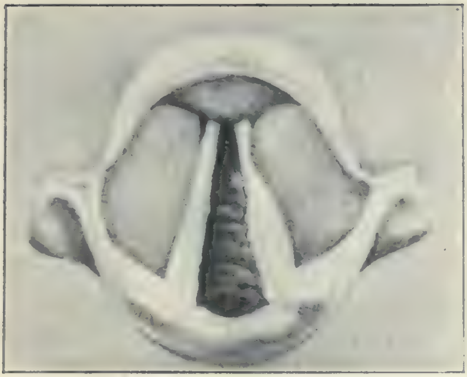

FIGURE 4.

J. A., aged twenty-four years. Sputum, positive. Pulmonary stage, second. Infiltration and erosion of both cords, especially left, with a longitudinal ulcer. Infiltration of both ventricular bands, partially covering true cords. Patient has marked hoarseness, and lesion, having passed an acute stage, is undergoing fibrosis. Diagnosis: Subacute tuberculosis of the larynx.

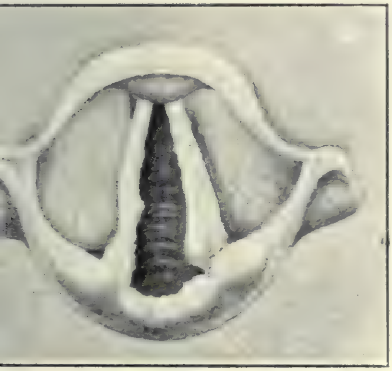

FIGURE 5.

ged twenty-three years. Sputum, posi-
lmonary stage, second. Marked infil-
nd ulceration of both cords, presenting
d-out appearance. Interarytenoid hy-
with wrinkling of mucous membrane.
dema of posterior portion of right ven-
band. Diagnosis: Subacute tubercu-
he larynx.

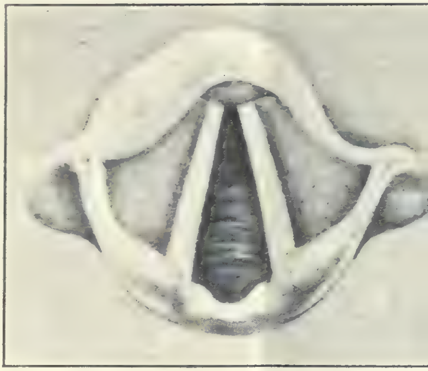

FIGURE 6.

M. A., aged twenty-seven years. Sp
positive. Pulmonary stage, third. Infiltr
limited to epiglottis, especially right side.
of larynx not affected. Patient has no ho
ness, no dysphagia. Occasional sensatio
fullness and tickling in larynx. Diagn
Subacute tuberculosis of the larynx.

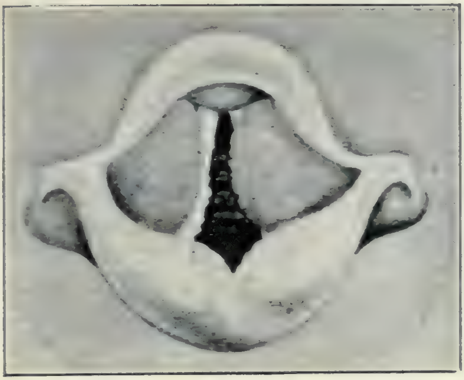

FIGURE 7.

P. R., aged thirty-one years. Sputum, positive. Pulmonary stage, third. Marked infiltration and ulceration of both arytenoids, presenting a pear-shaped appearance. Infiltration and ulceration of both true and false vocal bands, the latter almost entirely covering the true cords. Marked hoarseness, and sensation of fullness of larynx. Patient also gives a three plus Wassermann reaction. Diagnosis: Acute tuberculosis of the larynx, with a luetic infection as a probable underlying factor.

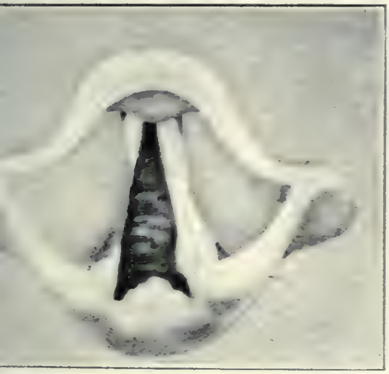

FIGURE 8.

aged twenty-eight years. Sputum, posi-
ulmonary stage, second. Tuberculoma
arytenoid space with healed erosion.
ing and infiltration of both cords, espe-
e left. Left cord also shows healed ul-
s on its upper surface. Infiltration of
ntricular bands, especially the right,
lmost entirely covers the right cord.
shows complete fibrosis, and outside of
hoarseness, patient has no discomfort.
is: Chronic tuberculosis of the larynx.

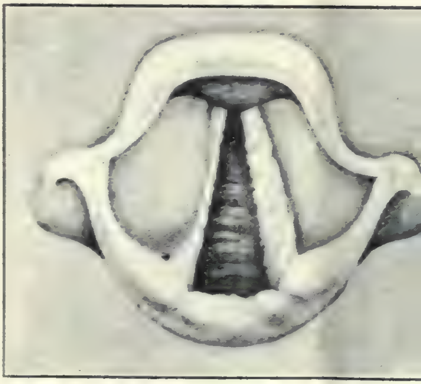

FIGURE 9.

L. F., aged forty-two years. Sputum,
tive. Pulmonary stage, second. Mark
filtration of both cords, especially the
Cords present a rough and granular a
ance. Marked infiltration of right ventr
band overlapping the true cord. Interary
hyperplasia with the characteristic "squa
of the interarytenoid space. Slight g
edema of the mucous membrane of the l
Hoarseness marked. Diagnosis: Subacu
berculosis of the larynx, with slight tende
fibrosis.

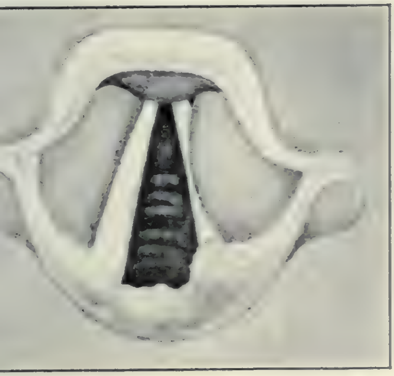

FIGURE 10.

aged twenty-one years. Sputum, posi-
lmonary stage, second. Marked infil-
nd thickening of left arytenoid cartilage
ventricular band, partially hiding the
l. Other findings negative. No hoarse-
iagnosis: Subacute tuberculosis of the
vith marked tendency to fibrosis.

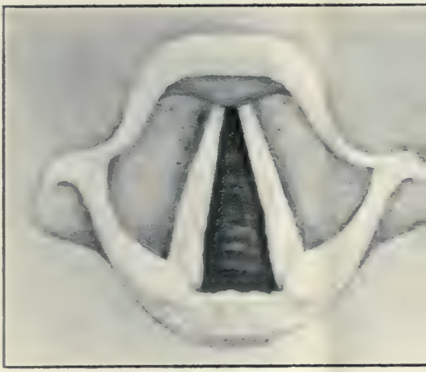

FIGURE 11.

H. H., aged twenty-one years. Sputum,
tive. Pulmonary stage, second. Distin
terarytenoid hyperplasia with lateral fur
Infiltration of both vocal processes. Note
acteristic "squaring" of posterior commi
(Casselberry.) Diagnosis: Chronic tul
losis of the larynx.

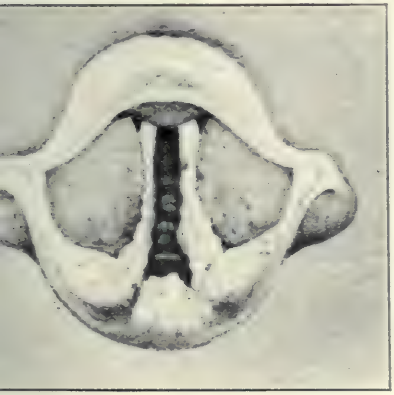

FIGURE 12.

aged twenty years. Sputum, positive.
ary stage, second. Marked infiltration
perficial erosion of epiglottis. Infiltra-
erosion of both true cords, ventricular
nd arytenoid. Marked interarytenoid
asia with erosions. Distinct lateral fur-
Patient has marked hoarseness and sense
ess in larynx. No dysphagia.

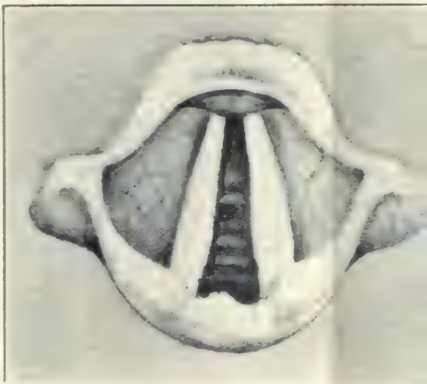

FIGURE 13.

Same as Fig. 12 after seven months of
ment. Edema almost entirely disappeared
cerations with the exception of interaryt
space all healed. Marked improvement in
nation. Lesion taking a more chronic c
and healing by fibrosis. Diagnosis: Acu
berculosis of the larynx, gradually healin
fibrosis.

XLVI.

THE RADICAL MASTOID OPERATION.*

By Thomas J. Harris, M. D.,

New York City.

In the year 1891, Stacke, at present professor of otology in Erfurt, brought forward an operation for the cure of chronic suppuration of the ear, which in its modified form is known the world over as the Schwartze-Stacke, or the radical operation on the ear. In the twenty-five years which have elapsed, the operation has been performed hundreds of times and by many operators. The present time, then, is a fitting one for an unbiased consideration of how far the operation may justly be regarded as accomplishing what it was designed for. We undertake this, not unmindful of the recent excellent paper on the same subject by Dr. S. MacCuen Smith.

To do this with any degree of satisfaction a correct understanding of two points is essential. For want of doing this, many a like inquiry has proved a failure. First, exactly what is the radical operation? Second, when is it indicated?

These two questions may be answered together in a single sentence, by saying that the radical operation, applied to the ear, means, as elsewhere in the body, an operation for the radical or complete removal of all disease, and is indicated when cure by other measures is found impossible.

The simple opening of the mastoid as devised by Schwartze had at the time that Stacke reported his operation been performed for eighteen years. While successful in many cases, especially in those of an acute nature, all operators found that it often failed to completely cure the disease when the suppurative process was a chronic one. To accomplish this,

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*Read before the Annual Meeting of the Section on Diseases of the Eye, Ear, Nose and Throat of the Medical Society of the State of New York, May, 1916, held in Saratoga Springs, New York.

Küster[1] in 1889 recommended a more thorough eradication of the disease by the removal, where necessary, of the posterior wall of the external auditory canal, together with the malleus and incus. Two years later, Stacke, with the same end in view, based on a series of twenty-five cases which he had performed, advised attacking the disease in the middle ear through the auditory canal, for this purpose exposed by the familiar postauricular incision. When this had been done, the antrum was opened through the aditus and all disease removed. In addition, if dead bone or granulations were found in the mastoid cells, these were opened and cleaned out. To cover the bony defect, a flap was recommended to be thrown

As now practiced, the radical operation has widely departed from the posterior wall of the membranous canal. from the original procedure of Stacke, and follows more closely the lines laid down by Küster, in that the antrum is opened in the usual way and then by an extensive lowering of the posterior canal, the mastoid cells and antrum, the middle ear and the external auditory canal are thrown together into one cavity, after thoroughly removing all disease.

It must be constantly borne in mind that the original indication for the operation, as laid down both by Küster and Stacke, was solely to cure a chronic suppuration. The fact that in the course of the operation intercranial complications are discovered, or of its performance as a means to relieve intercranial complications already discovered, must not be allowed to befog the simple and usual indications for its use.

Assuming that we are agreed now as to exactly what is the radical operation and what the true indication for its performance is, we are prepared to consider the main question before us—to what extent does the radical operation succeed in accomplishing what it is usually performed for? As far back as 1906, Dench, in a paper read before the American Laryngological, Rhinological and Otological Society (Transactions of the American Laryngological, Rhinological and Otological Society, 1906, page 258), reported his results to be seventy-five per cent of cures out of one hundred and seventy-three cases, the last ninety-five cases in this series showing eighty per cent of cures. Smith, in a paper read before the same society in 1915, reported eighty per cent of cures, which he claimed could be increased to ninety-five per cent or better in private work; "in thirty-two per cent of operated cases the hearing was definitely better, in forty-nine per cent relatively the same, and of the remaining nineteen per cent, in eleven per cent the hearing decreased after operation according to actual record, and in the balance, or eight per cent, it decreased according to the statement of the patient."

These two distinguished surgeons possess in the highest degree the technic essential to produce the best results. It must be recognized, however, that all operators, and indeed most operators, do not possess their degree of surgical skill, and our inquiry has to do with the results obtained not merely by a few, but by many operators. Are the results reported by Dench and Smith the results which are being secured by the rank and file of aural surgeons today? Stated succinctly, have we the right to expect from the results of the last twenty-five years that the radical operation will usually cure chronic suppuration; second, that it is a safe operation so far as injury to the hearing is concerned; third, that it is reasonably free from dangerous complications?

An authoritative answer to these three questions is difficult. It may be impossible. Statistics are proverbially misleading, and any one who has had occasion to consult hospital records knows how woefully lacking they are.

Straws, however, show the direction in which the wind blows. A short time ago the writer had an opportunity to follow the dressings in probably the largest of the foreign clinics. He was amazed to see the number of suppurating ears persisting after the radical operation, as well as the large number of facial palsies. The operations had been performed by half a dozen or less operators possessing the highest surgical skill. In this country he has been permitted to examine, in one of the leading special hospitals, all the records obtainable of the radical mastoid operations performed during the last two years. These numbered eighty-five. In most of them an accurate functional test before operation was lacking. Also, in most cases no statement appeared of the condition of the ear at the time of discharge from the hospital. Twenty-four patients responded to a postal card requesting them to call for examination. These cases were carefully examined, and the result of the examination is given under the four headings of

	Discharge	Hearing	Epidermization	Condition of Tube
1	Yes	Worse	Granulation	Granulation and on wall
2	Yes	V-10',W-2'		Open
3	No	?	Yes	Closed
4	No	?	Yes	Closed
5	No	?	Yes	Closed
6	Yes	Lost	Yes, except at open tube	
7	Yes, odor	Same	Still under treatment	
8	Yes	Worse	Partial	Open
9	No	Lost	Yes	Open
10	Yes	1' worse	Partial	Open
11	No	1'	Yes	Closed
12	No	None	Yes	Closed
13	Yes	2', better	Granulation inner wall	Open
14	No	Lost	Yes	Closed
15	No	Loud voice	Yes	Closed
16	Yes, odor	Same, 2'	Canal filled	
17	Yes, odor	Same, 2'	Under treatment	
18	Yes	Same	Granulation middle ear	Open
19	R. yes, slight*	1' better	Small post. wall	Closed
	L. No	2', worse	Yes	Closed
20	No	3'	Yes	Closed
21	Yes	1½'	Yes, except at open tube	
22	Yes, odor	2'	Except inner wall and open tube	
23	No	2'	Yes	Open
24	No	2'	Yes	Closed

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*Lecture Case

This summarized represents twenty-four cases with twenty-five ears operated upon. Of these, twelve ears were found upon examination to be perfectly dry, or forty-eight per cent. Thirteen, or fifty-two per cent, were still discharging. Of these, odor was detected in five. As regards epidermization, the ear was found fully epidermized in fourteen, partly in three, while granulations were found in five. Two cases were still under treatment. It may be added that in no instance had the operation been performed less than five months previous.

As regards hearing, except in one or two cases, no preoperative tests had been recorded. We were accordingly compelled to depend upon the statement of the patient as to the condition before operation. The Bárány noise producer was used in our examination in each case. Accurate statistics were found to be well-nigh impossible. The general impression gained from our questioning was that the hearing in the operated ear was for all ordinary purposes lost. Whether this condition obtained in most of the cases before operation it is impossible to state. Two patients, or eight per cent, reported the hearing improved; in four, or twenty per cent, it was reported as having remained the same; while in four, or twenty per cent, it was reported worse. Of the remaining fifteen, we were in doubt as to the condition in three cases, in four cases the whispered voice was heard at two feet, one at one foot, one at one and a half feet, and one at three feet. In all of these seven cases, only one of which heard at more than two feet, we can fairly regard the tests as showing the hearing lost, and in a general way, in spite of the lack of accurate statistics before operation, we are forced to the conclusion that the contention of those who claim that the hearing is improved or maintained by the operation, is not borne out.

In eleven of the twenty-five cases the eustachian tube was closed so far as could be determined. In nine cases it was open. In ten cases where the ear was found perfectly clean, the tube was permanently closed. In only two of the perfectly healed cases was the tube found open.

These statistics represent the work of approximately a dozen operators, all of them men with large experience. They do not differ, in our opinion, from the results obtained in other hospitals. The number of cases actually examined is not large. We cannot, however, with any degree of confidence believe that the remaining fifty-five cases would have given any different results. Moreover, from conversation with colleagues the country over, the findings are entirely in line with what in their experience exists.

Indeed, a careful analysis of the results reported by Dench indicates that what he regards as a cure differs from what we understand it to be, for he qualifies his statement of "80 per cent of cures" by stating that "in no instance did the discharge remain profuse after the operation, all of the cases being either dry or presenting but slight discharge from the meatus." Further on he refers to cured cases returning at intervals with the complaint of discharge from the canals. Exclusion of such cases would decidedly alter the figures which he gives.

With the limited and insufficient data before us we are forced to the conclusion, then, that the results so enthusiastically claimed by some of the advocates of the radical operation are not being uniformly secured. It would appear that approximately fifty per cent, or every other case, shows, after an interval of several months following operation, persistence of the discharge to a greater or less degree.

We were, morever, impressed in our study of the cases by the frequency with which patients volunteered the information that the discharge from the ear was no whit better following the operation. In a still greater percentage of cases the hearing in the operated ear was for all practical purposes lacking, although we have no accurate information to show in how many cases the hearing was unsatisfactory before operation. This aspect of the ear, particularly from its economic standpoint, was to us most distressing, and should give food for thought.

Is the operation then on this account to be condemned or given up? A thousand times no. The innumerable cases that it has cured is sufficient proof that it has come to stay. Such being the case, what are the reasons for the failures in so large a percentage of cases? In our opinion, the two chief reasons are: First, failure in determining the proper indications for the operation, and, second, faulty technic in the operation or in the postoperative treatment.

First, in regard to the indications. There is no question in our mind that in many instances the radical operation is unnecessarily performed. An important discussion on this subject took place before the American Otological Society in Boston, in 1907. At that meeting papers were read by Dr. Crockett of Boston and Dr. McKernon of New York, while the discussion which followed was engaged in by numerous aurists from all over the country. The whole trend of the discussion was in line with the assertion just made, namely, that the operation was being performed without a careful determination of the indications for its use. A suppurating ear extending over a few weeks or months, where the probe shows that no dead bone is present, is not a proper case for the radical operation. Such cases should be subjected to a careful and systematic course of treatment before any operation is to be considered. Especially is this true if the discharge is only intermittent and the hearing only slightly reduced. Proper drainage through incisions in the drum and unremitting care in keeping the nasopharynx clean, will perfect a cure in a large percentage of these cases.

The second group, where the radical mastoid is not required, as pointed out by McKernon, is composed of children with the history of discharge following the exanthemata. Here removal of the adenoids and tonsils with conservative treatment to the ear is the proper course to pursue. In this way the hearing is preserved. It is impossible in any of these cases to say how long local treatment should be kept up. The statement of McKernon is significant, that he has seen a number of cases in both hospital and private practice where after a radical operation the patient has volunteered the statement that if he had known the hearing would have become so poor he would never have had the operation performed.

With the risk of being regarded as an ultraconservative, we venture to say that the fetish that every suppurating ear is a volcano which may explode at any moment, is far from the truth, and is responsible for many an operation which otherwise would not be performed. Kipp, who at the time of his death had as large an experience as anyone in America, stated at the meeting just referred to that he had never seen intercranial complications follow ear trouble which was under treatment. While Kipp may have been more fortunate than most of us, the total number of such cases in any one man's practice cannot be large. Crockett is particularly emphatic in his advice against operating radically upon an ear with good hearing, in case the hearing in the other ear is destroyed, unless the indications are particularly exacting.

With patients with double chronic suppurative middle ear disease, he feels that only in the presence of symptoms indicating danger to life should the radical operation be performed.

Again, when conservative treatment has failed to stop the discharge, it is by no means certain that the complete or radical operation is necessary. In the words of Blake, "the all-important point is the determination of the main source of the suppurative discharge, whether it is antral or postantral, or mainly from the tympanum, including the carious process in the ossicles, or whether spontaneously ceasing and frequently recurrent, it has its source from reinfections in the eustachian tube."

Careful study of the case, including the aid given by a good X-ray picture, will generally show whether the disease is limited to the antrum and mastoid cells. If so, the classical Schwartze operation will prove sufficient. In this way the middle ear will be left undisturbed and the hearing unimpaired.

All aurists are agreed as to the important rôle that the eustachian tube plays in keeping up infection in the middle ear. Bryant goes so far as to assert that every case of suppuration from the middle ear can be cured by appropriate treatment addressed to the nasopharynx. Hays,[2] in a paper read before the New York Academy of Medicine in 1910, quotes as the opinion of a large number of men with whom he has talked, that eighty per cent of all cases of suppuration from the ear can be cured by appropriate measures directed to the tympanum, without resorting to the radical operation.

An all-important part of such treatment is an effective checking of the infection from the nasopharynx. This the favorable reports presented in the paper by Yankauer in Chicago last year would show can be secured by the closure of the tube according to his method. This operation has now been before the profession for six years. We at first were inclined to feel that the closure of the tube was not a scientific procedure. In the light of the statistics given by Yankauer, representing one hundred and nineteen operators and a total of seven hundred and thirty-five operations performed, in which six hundred and nine, or eighty-three per cent, of the cases presented a closed tube, and in fifty-one, or five-tenths per cent, the suppuration cured, makes us feel that the operation is of decided merit. Yankauer, indeed, is so optimistic that he feels that every case should be given the chance of a cure by closure of the tube before proceeding to the radical operation. While most of us are not prepared to follow him to that extent, it seems reasonable to assert that in cases where no necrosed bone is discoverable and the secretion is mucous rather than purulent, without odor, especially if the X-ray picture reveals little or no trouble in the mastoid cells, the operation should be performed, if only, as Yankauer suggests, as a preliminary step to the radical operation, which always demands the closure of the tube. Yankauer in his paper touches upon the all-important question of the effect of the operation upon the hearing. Concerning this he is equally encouraging, inasmuch as he states that in only four per cent of the cases was the hearing made worse, while in forty-six per cent of the cases it was reported as improved.

We are not enthusiastic in regard to the benefits of the conservative mastoid operation of Mr. Heath, but certainly his insistence upon the importance of an attempt to preserve the hearing and the neglect that aurists have shown in their operative work in this respect is deserving of the most careful consideration. It was clearly the intention and advice of Küster and Stacke that the radical operation should be performed only when the disease has been found to extend into the middle ear. If the operation is performed in this way, the criticism of Mr. Heath is not warrantable; but too often it is performed without any proper investigation of the exact situation of the disease. We fear the statement of Mr. Heath is only too true, that "with the exception of those patients the condition of whose ears had become so dangerous to life as to demand early operative assistance, the saving of the hearing has been purely a matter of good fortune."

To recapitulate, as the result of this investigation we are drawn to the following conclusions:

First, that the radical operation is an operation of undoubted merit.

Second, that it has been in the past and is today being performed often when not called for.

Third, that the results are by no means uniformly good, partial or complete failures occurring in a considerable percentage of cases.

Fourth, that improvement in the hearing cannot be promised. The most that can be offered, in the light of our statistics, is that the hearing will not be altered, although there is sufficient risk of lowering or destroying it to warrant reluctance or refusal to operate in case the hearing in the other ear is destroyed.

Fifth, that while accidents, including facial paralysis, are met with in the course of the operation, they are not of sufficient frequency or significance to have any bearing upon a decision in regard to the operation.

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XLVII.

ACCIDENTS IN BRONCHOSCOPY.*

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The subject of the direct method of examining the bronchi and the esophagus, together with the increase of the operative possibilities, now presents a broad field in the literature of laryngology; this, with the added great interest which has been shown in the subject by the profession in general, is the apology which I have to offer in attempting to add in a slight degree to this interesting study by presenting for your consideration a few accidents incident to our work.

An accident may be defined as an event that was unexpected, or the cause of which was unforeseen. My first case of bronchoscopy died as the result of an accident, when the patient seemed on the high road to recovery. With the time at my disposal the scope of the paper must be limited. An attempt has been made to recite a few personal experiences as well as a number of accidents I have been able to collect from the reports of other operators.

While increasing experience will diminish the possibilities of accidents in the future, we in this, as in all special fields in surgery, must expect the unforeseen to occur, and it seems remarkable to me, in a search through the literature, that so few accidents have been recorded.

It is wise for the reader in passing to remember in taking up the accidents in the operative use of the various instruments, that much might be added if we were to consider the fatalities incident to the nonuse of the bronchoscope.

No attempt has been made by Brüning, Jackson, Mosher or Johnston, in their recent books, to classify the accidents occurring in bronchoscopy and esophagoscopy. Johnston [1] quotes Kahler as having collected in the last two years two hundred and ninety-one cases of foreign bodies where only twenty-seven, or 9.6 per cent resulted in death; a considerable number he states were due to accidental circumstances.

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In the analysis of my cases I have arranged them under three divisions:

First.—Accidents during the anesthetic stage—that is, previous to actual instrumentation.

Second.—Accidents during the instrumentation, either for operation or examination.

Third.—Accidents occurring in the postoperative stage of our work.

Under the first heading may be grouped the following cases:

Case.[27]—Chloroform poisoning. Child, male, aged twenty months, was treated for laryngeal diphtheria. While in the contagious hospital the child coughed up a piece of a safety pin: X-ray showed part of the safety pin still in the left bronchus. He was transferred to a general hospital November 6, 1913. Preparation for operation; chloroform was administered by a member of the house staff, unfortunately not familiar with chloroform. In a short time attention was drawn to the child, who was found to be deeply under the anesthetic, which had been administered in great excess. No attempt was made to operate. The child died that evening, never regaining consciousness.

Case.[15]—Child, aged four years. Sudden dyspnea during administration of a general anesthetic, due to dislodging of foreign body from bronchus. Rapid tracheotomy; a seed three-eighths inch long was found lodged in larynx. Recovery.

Case.[28]—Accidental dislodging of foreign body incident to relaxation of general anesthesia. Girl, aged thirteen years. History of having swallowed a baby pin two days previous to operation. Anteroposterior radiograph, taken two hours before the operation, showed the object in the neck, lower end being on the level of the first tracheal ring. The pin was supposed to be in the esophagus. An attempt was made to palpate the same under general anesthesia; this proved negative. A nine-millimeter tube was passed into the esophagus; object was not found. It was thus considered the pin had passed into the stomach. While there had been no respiratory difficulty and no change noted in the voice, as a precaution a seven-millimeter tube was passed into the trachea and the object could be seen at the opening of the right main bronchus. It is interesting to note that the removal was easily accomplished, although the pin was open.

Case.[28]—Child, aged eighteen months. Metal disc at the upper end of the esophagus. Under the relaxation of a general anesthetic the object had disappeared when the tube was passed. On further introduction the object was found at the lower end of the esophagus, and was successfully removed.

ACCIDENTS AT THE TIME OF OPERATION OR EXAMINATION.

Case.[27]—Child, aged three years. Sudden cessation of respiration during lower bronchoscopy under ether anesthesia; death. A superior bronchoscopy was done for the removal of a foreign body; careful search negative. A lower tracheotomy was performed. On introducing bronchoscope through tracheotomy wound the child suddenly stopped breathing. Death. No postmortem.

Case.[7]—Sudden death during bronchscopy through a lower tracheal opening. During manipulation, before foreign body was seen (a tack in the bronchus), the child suddenly became cyanotic and died.

Case.[7]—Sudden death during bronchoscopy through a lower glands. Child died suddenly during the bronchoscopy; immediate removal of the tube, artificial respiration, and other efforts negative. Autopsy showed a mass of glands in the mediastinum, pressing on the trachea and left bronchus, almost obliterating the lumen of both. This case is hard to understand from the autopsy, unless the tube passed into the left bronchus, thus cutting off the right or good lung.

Case.[1]—Sudden collapse, recovery, girl, local anesthetic, bronchoscope in the trachea, spasm of chest muscles which entirely shut off respiration. To all appearances child was dead. Under treatment she slowly rallied, and was normal within an hour.

Case.[1]—Sudden collapse of adult under cocain and ether anesthesia occurring during esophagoscopy. As tube entered the esophagus patient stopped breathing. The operation was discontinued; verge of death; stimulation, artificial respiration; recovery at the end of several hours.

Case.[1]—Male, adult; local anesthesia; bronchoscope passed into the trachea. Patient became suddenly unconscious and cyanotic, falling off stool; acted as previous case; recovery. Operator was unable to explain the cause in either case.

Case.[4]—Foreign body, a bean, fell over into the healthy bronchus when extraction was attempted, and induced asphyxiation with fatal termination.

Case.[4]—Same.

Case.[4]—Same.

Case.[28]—Accident to lighting element of bronchoscope during operation for removal of pin from bronchus of child aged twenty-four months necessitated discontinuance of operation.

Case.[28]—Accidental loss of foreign body, pearly part of an oyster shell in the esophagus for three days; X-ray located object. On attempting removal through an eleven-millimeter tube in upright position, the object was grasped by forceps, but they failed to maintain their hold; the object passed slowly down the esophagus, eluding the grasp of the forceps, and finally escaped into the stomach; recovery in stool.

Case.[33]—Perforation of esophagus at time of operation. Attempts were made to remove a safety pin from the esophagus by use of a Jackson tube. The object was not seen, but could be felt; several attempts were made to remove the pin, using the forceps blindly. Operation not successful; death. Postmortem, small perforation found in wall of esophagus.

Case.[6]—Accidental change of position of foreign body at the time of operation; small grain of raw corn not found in lung, but on withdrawing the tube it was found just beneath one vocal cord. With the child's head lowered the foreign body had left its original position in bronchus and gravitated to upper end of trachea.

Case.[28]—Accidental opening of a periesophageal abscess in effort to find a fishbone swallowed some days before. Passage of instrument, the number nine-millimeter Kahler tube, discovered small edematous area about one inch below cricoid cartilage. Object not found, but a sudden gush of pus proved that I had to deal with a small periesophageal abscess which had opened spontaneously. Recovery.

Case.[36]—Sudden flooding of bronchi in attempt to remove pin from the bronchus, due to opening a pulmonary abscess. Later lower bronchoscopy and successful removal.

Case.[37]—Pneumothorax developed during the attempts to remove hook by superior bronchscopy; removed subsequently by inferior bronchoscopy. Recovery of object and patient.

Case.[38]—Child, aged seven years. Sudden death during superior bronchoscopy, due to debilitated condition and prolonged manipulation, and not due to anesthesia. Autopsy.

Case.[5]—Accidental breaking of forceps. Failure to remove pin from left bronchus, so abandoned operation. At this operation the miniature lamp and a small piece of metal became detached and fell into the bronchus.

Case.[30]—Accidental slipping of forceps as foreign body reached the larynx; cyanosis; emergency tracheotomy. Late recovery of ring by direct laryngoscopy, the ring having remained in the subglottic region.

Case.[31]—Arrest of respiration during superior bronchoscopy. Rapid tracheotomy and successful inferior bronchoscopy.

Case.[32]—Bean dropped into opposite bronchi during extraction by superior bronchoscopy, again grasped and removed.

Case.[34]—Severe dyspnea during superior bronchoscopy necessitating rapid tracheotomy.

Case.[35]—Severe spasms and cyanosis during superior bronchoscopy for removal of bean. Rapid tracheotomy. Recovery.

Case.[4]—Asphyxiation during attempts at removal of a marble bead. Rapid tracheotomy. Recovery.

Several cases.[4]—Foreign body too large to extract through larynx. Note.—Cases of this type might be included.

Case.[9]—Woman, aged thirty-six years. Interesting accident. During attempt to remove a plum stone from right bronchus the forceps slipped at larynx and the stone dropped into left bronchus. It was seen and grasped but could not be removed, and the patient died in a few hours. Postmortem. The stone was found in left upper lobe of bronchus firmly imbedded. Paterson [17] quotes Brunning as suggesting the use of a bronchial protector to cover opposite bronchus before attempting removal of this type of foreign body.

Case.[9]—Boy, aged fourteen years. Death from severe hemorrhage at the time of operation. Button imbedded in granulation tissue in the left main bronchus. Attempt to remove it with blunt pointed tenaculum through bronchoscopy tube caused severe bleeding. Patient had coughing fit followed by an enormous wave of bright red blood from a pulmonary vein. Patient died promptly from exsanguination.

Case.[8]—Accidental perforation of the esophagus during an esophagoscopic examination made by an experienced surgeon and with due care. Perforation of esophagus was found at autopsy above a cancerous growth. A retroesophageal abscess had developed and patient died of inhalation pneumonia.

Case.[2]—Kummell, 1913.—Accidental perforation of esophagus in two cases, not with the esophagoscope, but with the preliminary introduction of the bougie at the time of the operation. In both cases the mishap proved fatal. In both the lack of subjective disturbance at the time and the absence of bleeding as seen by the esophagoscope are noted; abscess developed outside the esophogus (might have been saved by opening). It is of interest to note that no later than 1912 an American laryngologist recommends in passing a tube for direct examination that it is desirable to first locate the position of the body by a bougie and to use an obturator in passing the esophagus.

Case.—(B)—Slipping of forceps in recovering tack from bronchus. Lost—further search negative. Tube withdrawn; tack found almost immediately in mouth.

Case.—(B.)—Another case same.

Case (B)—Slipping of the forceps, loss of the tack. Unable to find; operation abandoned. New radiograph showed the tack in opposite upper bronchus; removed, having secured stronger forceps.

Case.[4]—Emphysema skin; phlegmon and fatal mediastinitis from accidental pricking of trachea during removal of cigar tip from bronchus.

Case.[16]—Slipping of forceps, releasing foreign body at the time of removing. Remained impacted in subglottic space and easily removed by the crocodile forceps.

Case.[39]—Child, aged seventeen months. Sudden death twelve hours after removal of jackstone from esophagus by esophagoscopy. Anesthetic. No postmortem. Death ascribed to either shock or subglottic edema.

Case.[40]—Pulmonary abscess, death. Child; bronchoscopy. Removal piece of fig. Child developed pulmonary abscess. Death; cause, incomplete removal of foreign body.

Case.[40]—Shock; child. Bronchoscopy for the removal of a peanut; successful. Case was almost moribund at the time of operation. Died within a few hours.

Case.[41]—Male, aged forty-six years. Patient died one week after direct laryngoscopy. Only case of death in operator's practice from general anesthetic. Gangrenous bronchitis due to delayed chloroform poisoning.

Case.[41]—Child, aged four years. Death. Rhythmic convulsions three hours after direct laryngoscopy. Idiosyncrasy to cocain.

PRECAUTIONS.

The longer we work with delicately constructed instruments in the still more delicate living tissues of the bronchi and lung and the esophagus, the more fully do we realize the statement attributed to Dr. Killian,[2] namely, that the manipulation of foreign bodies through the esophagoscope was not everybody's business. This point is emphasized by Jackson,[10] who in replies from three hundred and eighty American laryngologists found that only thirty-five cared to do the work, and only seventy-nine had attempted the same. We are all anxious for the work to progress on scientific and safe lines, and it is interesting to note the enthusiasm which is shown by the men taking up this work in one of the New York postgraduate schools where a course is given by me in the principles and preliminary steps in the use of the bronchoscopic instruments. These men realize they have only begun their work in a department of laryngology in which the field is broad and the technic precise and progressive.

Before taking up our analysis of the cases, we must assume that bronchoscopy is a hospital operation, and the earlier we see the case the better the possibilities of our work, and the better prognosis to the patient.

In our first group of cases the difficulties of anesthesia are to be overcome. In these days of specialists in anesthesia the question of the administration of a general anesthetic in our larger cities can be easily met. It has been my custom to try to secure the services of one of these men to relieve me of the responsibility of this part of the operation, and those who have seen the work of Dr. Gwathmey in his administration of the anesthetic by the rectal method, will realize the aid which it can be to the operator. Time will not permit me to take up this subject more fully.

Mention must be made to emphasize the value of oxygen in combination with ether or chloroform in cases with the slightest degree of dyspnea, and a plea for its use from the beginning, anticipating rather than awaiting the development of trouble.

To those who advocate and are able to work without the use of an anesthetic the question is easily solved. It is not my experience, nor have I witnessed any success in children, of attempts to examine the larynx and trachea without the use of an anesthetic. In one case I was successful in using Dr. Jackson's long spatula for the removal of a coin from the upper part of the esophagus in a baby without the use of an anesthetic, but then only with the combined assistance of three medical men. Up to this point I have felt that my work could be more easily accomplished and with greater safety to the patient, if I had the assistance of a skilled anesthetist. I have had only one fatal case that could be attributed to the use of the anesthetic. If there be a risk it is regrettable that the cases have not been reported, for we do not desire to exaggerate the dangers of the general anesthesia in this special branch of surgery, since in other operations we are so strongly urging the use of anesthesia for the relief of pain as well as mental effect on the future of the child.[10] It is suggested that in use of general anesthesia the cough reflex should not be abolished, and that we may use general anesthesia in children when a sharp body threatens perforation. The drawback to a general anesthesia, as given by one writer,[10] is a risk of respiratory arrest when the operator begins to manipulate the foreign body. In our first subdivision of cases we have one death from anesthesia. One case of sudden dyspnea due to dislodgement of foreign body, with recovery. Two cases in which change of position of foreign body was undoubtedly due to relaxation of a general anesthetic. The death can be attributed to the lack of a trained anesthetist. I see no way to avoid the accidents in the three other cases of change of position of the foreign body, and still feel that the struggle incident to the nonuse of a general anesthetic more than counterbalances the possible harm.

Under the second division we find the following cases reported:

Sudden death.—One case (cause obscure).
Sudden dyspnea.—Seven cases, three deaths.
Collapse.—Three cases, no deaths.
Change of position of foreign body.—Ten cases, four deaths.
Accident to lighting element.—Two cases.
Accidental slipping of forceps.—Four cases.
Opening periesophageal abscess.—One case.
Opening pulmonary abscess.—One case.
Added foreign body from broken instrument.—One case.
Pneumothorax.—One case.
Hemorrhage.—One case, one death.
Perforation of esophagus.—Three cases, three deaths.
Emphysema traumatic.—One case, one death.

It is of interest to note that the case of sudden death on the table could only be accounted for by the debilitated condition of the patient, plus the prolonged manipulation. The author distinctly stated that the anesthetic was not at fault. Sudden dyspnea with seven cases and three deaths seem hard to explain; of the two deaths, one at least would seem to coincide with the period of the operation considered dangerous by Dr. Jackson when performed under general anesthesia—namely, respiratory arrest when the operator begins to manipulate the foreign body. Three cases of collapse are noted. Mosher speaks of a cardiac arrest, reflex in type. Johnston's two cases of collapse were under cocain anesthesia. The change of position of the body during attempts at removal could be accounted for by the forceps losing their grasp and the object slipping. The construction of the forceps and the numerous new tips which have appeared will no doubt lessen this accident in the future.

Of the three deaths due to perforation of the esophagus, one was a pure accident, the two others were accidents which I am sure would not occur today, the lesson having been learned by the bronchoscopist,[13] [14] and the grave dangers to the preliminary probing to locate foreign bodies and the use of the obturator in introduction of the esophagoscope are now fully recognized. The golden rule is to keep your eye on the presenting body at the end of your tube from start to finish.

It is well to again warn the general practitioner and the surgeon who still persists in the blind extraction of foreign bodies.

I have not found any cases of acidental death due to short circuiting, from use of rheostat and commercial current.

Under class three we note the following:

Edema larynx.—Ten cases, two deaths.
Pneumonia.—Five cases, five deaths.
Periesophageal abscess.—One case, no death.
Pneumothorax.—One case, no death.
Pulmonary abscess.—Two cases, two deaths.
Hemorrhage.—Four cases, three deaths.
Right hemiplegia.—One case, no death.
Thymus.—One case, one death.
Hysteric edema.—One case, no death.
Convulsions and paralysis.—One case, one death.
Shock.—Three cases, three deaths.
Chloroform poisoning.—One case, one death.
Cocain poisoning.—One case, one death.

The most frequent accident in our postoperative cases is edema of the larynx. Ten cases with two deaths. Kummell[3] even favors a tracheal opening under five or six years to avoid subglottic edema. It is well to note here the word of warning given by Jackson,[26] that postoperative edema in children must not be ascribed to edema until the bronchoscope has been passed to drain the mucus. He reports that children become dyspneic within twenty-four to thirty-six hours, not from subglottic edema, but from an accumulation of bronchial secretions relieved by second passing of the bronchoscope and the removal of a large quantity of secretion. Try this first, especially in young children, before doing a tracheotomy, but never wait.[26] In a dyspneic patient tracheotomy should be done early, never late. Other precautions are use of small tube, avoidance of trauma on introduction and pressure during manipulation of tube. One case is noted where tube size number nine millimeters was used in a child. This of course might justly be assigned to a faulty technic. Two deaths from anesthetic, one chloroform, one cocain, the operator tells us would not happen at present, owing to added precaution taken by him.

Pneumonia is next in frequency, five cases with five deaths. This suggests more caution in use of anesthesia, avoidance of prolonged manipulation and care to avoid trauma. Elevation of foot of bed helps to drain the bronchi, especially in young children. The three cases of death from shock seemed to have been unavoidable. The skill and experience of the operator must be taken into consideration in these cases of shock as well as the other accidents cited. It is reported[25] that the greater percentage of fatalities in this work occurred in cases where operator's total number of cases were less than three.

CLASS OF ACCIDENTS TO OPERATOR.

Before closing I would ask you to note one case reported in which a cocain solution being used in a bronchoscopic examination was coughed into the eye of the operator, causing a paralysis of accommodation. The second case, one of my own, is interesting; for the pin, a sharp steel lace pin, was coughed out through a bronchoscopy tube with the force of a dart from an air rifle, hitting the lens of my glasses with considerable force. Both cases emphasize the necessity of protecting the eye in this work.

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XLVIII.

A FACTOR IN THE PROGNOSIS OF ACUTE SUPPURATION OF THE MIDDLE EAR.*

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We have been taught that the prognosis of acute suppuration of the middle ear depends upon the powers of resistance of the patient, the severity of the infection, and the time at which drainage occurs, either spontaneously or as the result of paracentesis of the tympanic membrane, but it is the purpose of this paper to show that there is another factor which influences largely the termination of this disorder, and one that has not been given the consideration which it deserves, namely, the differences which may exist in the anatomic structure of the mastoid cells.

Examination of a considerable number of mastoid bones will reveal a great variation in the size, number and arrangement of the pneumatic cells in different specimens, in some instances the cells being large, in others small, some mixed, with large cells posteriorly, in others a few large cells adjacent to the antrum and in the tip, while in others the posterior portion contains few or many small cells with walls of variable thickness.

The cortex, also, is thicker in some instances than in others, while the extent of the cellular structure seems to vary according to the type of skull from which the bone examined is taken.

In brachycephalic skulls the cells do not extend, as a rule, as far posteriorly as in dolichocephalic skulls, while the sigmoid sinus is apt to be found somewhat nearer the meatus than we usually find it in the latter type. It is, consequently, worth while noting the type of skull before proceeding to operate on a patient for mastoiditis, as the slightest hint as to the location of the sinus is of value. These differences are marked and constant, and a little observation will enable one to readily determine the type of head from which a given mastoid comes.

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*Read before the Pan-American Medical Congress, San Francisco, June, 1915.

It is of interest to know in what manner different formations of cell structure can influence the course and termination of an infection in these parts, and in order to determine this it is necessary to briefly consider the pathology of mastoid inflammation as a whole.

It should be kept in mind that the mastoid cells are lined with a membrane similar to that lining the cavity of the middle ear, and a suppurative inflammation of the tympanum can extend by continuity, and also by the infection of cells distant from this point by contact of pus which has been forced by pressure or gravitated through the aditus and antrum into these relatively distant parts.

In mastoiditis we are not dealing with a true caries, but with a rarefying ostitis which has a definite and extremely interesting pathology.

As soon as infection of the cell lining occurs the membrane swells to from ten to eighty times its ordinary thickness, and, becoming lobulated, presents the form of true granulation tissue, while the interstices are filled with bacteria-laden pus.

The cell wall beneath the membrane now becomes studded with osteoclasts which gradually erode the bone until two or more cells are converted into one, and this process continues to the point where the inner or outer table have been perforated. There is now a reversal of this condition. As soon as perforation occurs the osteoclasts disappear and their place is taken by osteoblasts that build up a substance which finally becomes bone of the hardest variety. We know that the greater the relative surface exposed to infection, the greater is its power of resistance, as there is more surface from which may emerge the germ destroying leucocytes, and when we consider the fact that the surface of a sphere increases as the square of the diameter and the contents by the cube, it is obvious that the smaller the cell is the greater is its resisting power.

Continuous observation of cases operated on, both as to the nature and virulence of the infection, and as to the size of the cells, shows conclusively that the above theoretic proposition is borne out in clinical experience.

The question now arises, how are we to determine in advance the nature and extent of the cellular structures which are or may soon be invaded?

It is my practice in every case of suppuration of the middle ear to ascertain the nature of the infecting agent, to note the type of skull in the patient under consideration, and to have a skiagraph made of the mastoid bone on the affected side.

In some instances the latter is not easy of accomplishment, but an expert skiagrapher can nearly always secure a plate sufficiently clear for all practical purposes.

Having, then, secured the picture of the cellular structure of the mastoid, and knowing the variety of pathogenic bacteria responsible for the inflammatory state, it is not difficult to prognosticate with reasonable success the probabilities as to whether or not mastoiditis will supervene.

Given a streptococcic infection and large cells in the mastoid, it is self-evident that the chances for such an unfortunate complication would be far greater, all else being equal, than where the infection was of the influenza bacillus and the cells small and of uniform size in their tendency.

The following case reports will serve as illustrations of the propositions set forth:

Miss L., aged twenty-six years. Previous health good. First symptom of aural difficulty, pain in the right ear, with great impairment of hearing, followed in about one hour by a profuse discharge of pus. Fourteen hours later, symptoms of like character began in the left ear, followed by a like discharge of pus in a little over three-quarters of an hour. Bacteriologic examination of the discharging pus showed a pure culture of streptococci in each ear. Type of skull, brachycephalic. The skiagraph showed absolutely no mastoid cells whatsoever. Suppurative process ceased in both ears inside of a week, with no noticeable impairment of hearing.

Deductions as follows: This patient probably had a small antrum and few, if any, pneumatic cells. The limited capacity of the bone for the storing up of pus caused rupture of the tympanic membranes from hydrostatic pressure at an extremely early period for an adult, while the nature and extent of the cellular structure presented the highest possible powers of resistance to the infecting agent.

Mr. R., aged thirty-eight years. Following an attack of influenza, he had pain in left ear for two days, with mastoid tenderness on the third day. First seen on the fourth day. At this time the patient's hearing in the affected ear was nil. Examination showed the tympanic membrane nearly normal in appearance and no bulging of the canal. Temperature 103° F. and perspiring freely during sleep. The mastoid tip was so tender it could not be even touched without causing great pain, and the patient complained of intense throbbing headache. Type of skull, brachycephalic. Blood count showed leucocytosis of 14,000 and neutrophiles 83 per cent. On the fifth day patient was sent to hospital and the mastoid opened. There were two extremely large cells below the antrum, the lower one occupying the entire tip and completely filled with pus and some granulation tissue. There was little pus and no granulation tissue in the cell above, and but few other cells found, and these were healthy. Bacteriologic examination of the pus found in the cells showed influenza bacilli and pneumococci.

Deductions: Large, freely communicating cells of small resistance and a rather severe infection. The size of the cells enabled them to act as a reservoir for the storage of pus, and the lower one in the tip became speedily infected. An unusual feature of this case is the lack of pronounced middle ear symptoms. Patient's hearing has never been better than ten-thirtieths since his recovery.

Mr. M., aged forty-one years. Following influenza he had severe pain in the right ear for six days, when the ear discharged spontaneously. I saw him first three days after this. Patient complained of violent headache, intense pain in the ear and weakness. Examination showed a large perforation of the drum head, but no bulging of the canal and no tenderness of the mastoid. Type of skull, dolichocephalic. Blood count revealed slight leucocytosis and polynuclear count of 79 per cent. X-ray showed a great number of small cells, nearly uniform in size, extending far posteriorly. Infection, influenza bacilli. Patient gradually developed symptoms of mastoiditis, but refused operation. However, on account of the persistent headaches and fear of permanent loss of hearing, he finally consented, ten weeks after the onset of the middle ear symptoms, to have the mastoid operation performed, as advised.

A great number of cells were found, filled with granulation tissue, but no pus.

Deductions: Mild infection, with small cells of great resisting power, enabled the patient to go on for a long time with comparatively mild symptoms. There is a possibility that this case might have terminated in resolution, if not operated on, but I doubt if his hearing, which is now normal, would have been as good.

The recitation of such experiences could be extended, as my observations along this line have been carried out for some years, but the ones selected to report are so typical and so representative of the whole number observed, that I do not feel it necessary to continue the report in further detail.

I assume, therefore, from the premises and the clinical phenomena observed, that anatomic variations in the cellular structure of the mastoid bone of different individuals influence to a marked degree the occurrence or avoidance of mastoiditis as a complication of acute suppuration of the middle ear.

Nor does the advantage of our knowledge of these facts stop here, for I venture to assert that such observations aid us in being able to conscientiously advise, at an earlier period than we otherwise could, in many cases, the performance of the mastoid operation.

XLIX.

A STUDY OF THE PHYSICOMECHANICAL FUNCTION OF THE FAUCIAL TONSIL.*

BY

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CHICAGO,

The study described in this monograph was undertaken for the purpose of throwing light on certain observations and certain quandaries which had arisen in the course of the practice of the authors. They had come gradually to realize that the appearance of tonsillectomized throats, as observed from time to time, had often indicated anything but ideal anatomic results, and in certain instances had pointed to unmistakable impairment of the depressor palatal muscles. A few cases had been met with in which the speaking voice had, following tonsillectomy, been seriously and permanently impaired. A few published reports had mentioned impairment of the singing voice as well. The articles of Dr. G. Hudson Makuen and Dr. Thos. R. French had made an impression. And perhaps more than all else, a feeling that the exclusively radical attitude of the profession with reference to operation on the tonsils of children, in view of the fact that the danger from such operation to the possible later development of the voice for artistic purposes remained as yet unsettled, was at least open to serious question. It seemed to them that the parents of the children and the children themselves possessed an inherent right to expect from the medical profession who cared for the children, in any procedure carried out for the child's health, certainly full consideration of a function which might later turn out to be of inestimable value to the child, and of great importance to society as a whole.

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*Presented to the Chicago Laryngological and Otological Society on March 21, 1916.

Note.—In this monograph the term "tonsil" will be employed to designate lymphatic tissue and capsule together, as a single organ.

Earlier in the history of the operation of tonsillectomy, one of the authors of this article had expressed himself as believing that the preservation of the function of the depressor palatal muscles, and so of the capabilities of voice development following tonsillectomy, was dependent upon carefulness and delicacy of technic in the performance of the operation. But in spite of the recent great advances made in respect to technic, the deformities of the throat following tonsillectomy are giving no certain indication of diminishing. While undoubtedly they may be aggravated by crudeness of technic, or by lack of operative skill, yet these deformities evidently are preventable neither by manner of operation nor by delicacy of technic. Dr. Makuen has forcibly pointed out cases of deformity of serious importance occurring in the hands of the most skillful operators.[1] The authors of this article can emphasize these observations from cases which they also have studied. In individual instances one may, by studying the end results of operations which he knows to have been done with great care and with great delicacy as to technic, witness a most surprising resulting picture of throat deformity.

In the systematic examination of tonsillectomized throats, one has it forced upon him that in most instances something has resulted to the throat besides the mere removal of a tonsillar mass. Not in all, but in a very large proportion, there is presented quite an altered anatomic picture. The relative regularity of this picture of deformity is of itself alone a most striking fact, and sufficient of itself to indicate the probable existence of a definite function of the tonsil with reference to the lower palatal muscles. If such a definite function exists, one would expect the deformities which resulted from total extirpation of the tonsil to present a certain tendency to uniformity in the manner of impairment of the action of the palatoglossus and palatopharyngeus muscles, and this uniformity to be directly traceable to the deprival of these muscles of a definite accomplishment with reference to their action performed previous to operation by the removed tonsil.

The authors are well aware of the incompleteness of the following study. They especially regret that up to this time they have not been able to obtain a cadaver upon which the anatomic results of tonsillectomy could be studied. But in spite of incompleteness they feel warranted in presenting their findings at this time, if for no other reason than that others may be encouraged to take up the subject for further study.

The manner of presentation of the matter in hand will be to discuss, first, the relationship of the tonsil to each of the depressor palatal muscles as shown by anatomic study; second, to point out the presumptive effect of depriving either muscle of the aid of the tonsil, and, third, to tabulate the corresponding facts as observed on tonsillectomized throats.

In the throats studied anatomically, we find only minor variations in the relation of the tonsils to the pillar muscles. Whether important variations may at times occur would require dissection of many throats. We have dissected five tonsils. From the standpoint of the investigation in question the extirpated tonsil is deceptive, since such a tonsil, if its capsule and mucous membrane covering remain intact, is by the action of these coverings bunched into a ball-like mass. In the throat, however, the border of what may well be called the floor of the tonsil—i. e., that part which lies against the lateral wall—tends to flatness, and may be tucked into narrow places where narrow padding is required. This floor presents definite border limitations, excepting below, where it becomes a part of the fascia bearing that part of Waldeyer's ring of lymphatic tissue which passes downwards into the laryngopharynx. Above and anteriorly the curved border of the floor parallels essentially the palatoglossus muscle. Following the border from below upwards, it lies between the palatoglossus and the superior constrictor, but drops below the palatoglossus as the latter approaches the soft palate.

The border curves downward at the palate and then parallels the edge of the palatopharyngeus at a short distance from this edge until the muscle enters the body of the pharyngeal wall. The attachments of the capsule to the palatopharyngeus are relatively short, and as one passes downwards into the laryngopharynx they become so short that separation is often impossible without bringing away muscle fiber. The floor of the tonsil spreads itself out over the superior constrictor, from which it is separated by a loose areolar tissue. The relation of the tonsil to this tissue is one of contiguity rather than of continuity; and yet, in one tonsil of the five examined, a thin, broad plate of connective tissue passes downwards along the inner surface of the superior constrictor and, overlapping the outer surface of the capsule of the tonsil, merges into its structure. In that one case the impression is directly given that the tonsillar capsule constitutes an elaboration of the intrapharyngeal aponeurosis, as is believed by Descomps, Patterson, Makuen and others. The impression is fully confirmed as one follows the capsule into the laryngopharynx, where its direct continuation unquestionably does become the intrapharyngeal aponeurosis. Removal of the capsule exposes the bared surfaces of palatoglossus and palatopharyngeus muscles, and often also of the superior constrictor, exactly as muscles are exposed more posteriorly and lower down by removal of the aponuerosis. Moreover, the elaboration of the tonsil into a structure having for its purpose the direction and protection of muscle structures, as shall be seen later, is characteristic of intermuscular connective tissue everywhere in the body. And yet, considering lymphatic tissue and capsule together, this elaboration has become so complex and purposeful as to constitute an organ of itself, a combined connective tissue—lymphatic tissue, pad-like organ, for certain definite purposes having to do on its physical side with the complicated muscular movements of this region.*

RELATION OF THE TONSIL TO THE PALATOGLOSSUS MUSCLE.

The palatoglossus muscle is in the cases examined small and relatively delicate, although, according to Dr. Makuen,[2] it becomes large in singers. It passes in a slightly outwardly bowed course from the side of the soft palate, downwards and forwards to its insertion at the side of the posterior part of the mouth portion of the tongue. Its position at the side of the throat is chiefly determined by its close attachment throughout its course to the under surface of a bridge of mucous membrane constituting the covering of the anterior pillar. The bridge-like character of this layer of mucous membrane is due to its passage over from the lateral wall of the throat onto the surface of the tonsil, where it either merges indistinguishably into the mucous membrane covering the surface of the tonsil, or else ends in a fairly well defined border on the surface of that mucous membrane. (Figure I.) It is to be carefully noted that this bridge of mucous membrane depends on the tonsil for its inner support. (Figure II, A, B.) Dissecting up this bridge of mucous membrane one finds the palatoglossus closely attached to its under surface. Between the palatoglossus and the inner surface of the lateral wall is a small irregular unoccupied space, bounded below by the upper anterior border of the capsule of the tonsil, and above by the bridge of (removed) mucous membrane. To the outside is a loose fascia covering the inner surface of the superior constrictor. The anterosuperior border of the capsule (with its accompanying lymphatic tissue) presses upwards from below into this bridged space so as to be, to a greater or lesser degree, between the palatoglossus and the superior constrictor muscles. It is attached to the palatoglossus quite closely by short strands of connective tissue, while to the superior constrictor the connecting bands are longer. Thus the palatoglossus muscle is encased in a canal determined by the superior constrictor, the capsule of the tonsil and the mucous membrane bridge. Its direction of action is governed chiefly by the outward attachment of the bridge of mucous membrane, which prevents the muscle to a certain degree from straightening towards the lumen of the throat as it becomes shortened in action. The upper anterior border of the tonsil serves to protect the muscle from direct contact with the superior constrictor, especially during the relatively violent movements of swallowing and retching.

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*In this article, for reasons of simplicity, we have not attempted to differentiate the relative part played by the capsule of the tonsil and the lymphatic tissue in the physical purposes accomplished by the tonsil.

As one approaches the soft palate, since the muscle approaches more closely to the lateral wall of the mouth, this bridge of mucous membrane becomes narrower. Here the capsule of the tonsil does not rise so high as to lie between the palatoglossus and the lateral wall.

The dependence of the palatoglossus on the tonsil may be understood when we consider the situation of the muscle with the tonsil removed. (Figure III, A, B.) Not only has the inner support to the bridge of mucous membrane been removed, but with it also the border of the tonsil which separated the palatoglossus from the superior constrictor muscle. The layer of mucous membrane supporting the palatoglossus has now, while still bearing the muscle, been transformed into an overhanging ledge. Nature's delicately complex plan for obtaining a perfect action of the muscle has been rudely undermined. The muscle is now in much the same predicament as a ship with a hopelessly injured rudder in a stormy sea and lying in the midst of a jagged rocky shore. The muscle, like the ship, is destined to be seriously shaken at the best, since its perfection of action cannot be hoped for excepting under essentially natural aiding conditions. The degree of the impairment of its function will depend on the strength of the mucous membrane ledge, on the extent of the dropping of the ledge from its former position, on the formation of scar tissue in and upon the muscle, and on the place and extent of adhesions formed between the muscle or ledge and the outer wall.

With the expanding effect of the tonsil removed, the breadth and outward curve of the superior constrictor diminishes (a process which is aided by the contraction of scar tissue) ; thus the perfection of action of the superior constrictor must be at any rate to some degree impaired. If the ledge of mucous membrane be both narrow and stiff, and if it lie in a favorable direction, these conditions may possibly save the muscle from the rocks of adhesion against the outer wall, even though the muscle cannot be saved from the inevitable scar tissue contraction and the retraction resulting from the almost inevitable sagging of the ledge. (Figure IV, A, B, C.)

By reason of the lack of supportive capability of the ledge of mucous membrane, or by reason of reparative or inflammatory swelling following the operation of tonsillectomy, or by reason of the traumatisms resulting from the violence of the movements of swallowing and retching, or by reason of these influences combined, contact between this mucous membrane ledge and the outer wall is apt to result in permanent adhesion. This adhesion might be slight or heavy, might extend over a small portion of the linear coures of the ledge, either anterior or posterior, or might be complete, in which last case the palatoglossus would be lost in an adherent linear scar throughout its entire course. In case of partial adhesion a part of function of the muscle would be retained. Adhesion posteriorly in the vicinity of the soft palate would be more serious to the function of the palatoglossus than adhesion anteriorly or even near the middle of its course. If adhesion occurred to the palate, and should a part of the function of the muscle be retained, nevertheless the direction of even the partial action remaining would be so extremely altered outwardly as to be relatively ineffective; and, even worse, if resulting scar tissue pulled too hard, the neighboring palatopharyngeus might be pulled with the soft palate over against the outer wall. (Figures V, VI, VII, VIII, IX.)

THE RELATION OF THE TONSIL TO THE PALATOPHARYNGEUS MUSCLE.

The exit of the palatopharyngeus muscle from the soft palate occurs in close vicinity to, indeed is nearly identical with, the place of exit of the palatoglossus; but the two muscles upon emerging from the palate begin at once to diverge the one from the other. The size of the angle of divergence of these muscles is of considerable importance and varies approximately from thirty to fifty degrees. The palatopharyngeus is, relatively to the palatoglossus, a much larger and stronger muscle. It is flattened anteroposteriorly to a fan-like shape, and, extending at its lateral borders to the outer wall, serves as a continuation of the general surface of the soft palate downwards on each side. It passes downwards and backwards to a point opposite the base of the tongue, where it merges into the general muscular mass coursing along the anterolateral surface of the pharynx. In this study we shall not attempt to follow the muscle in its lower pharyngeal and laryngeal relations.

The tonsil appears to serve the palatopharyngeus in two important respects. In order to appreciate the first of these one should realize the thinness and feebleness of the bared curtain of muscle, as well as the fact that the lateral attachments of the muscle are fixed, while its central attachments in the soft palate are to a certain degree movable. The capsule of the tonsil lies against the lateral wall and curving inwards lies also against the anterior surface of the palatopharyngeus muscle. (Figure X.) Thus the muscle is, as it were, braced with reference to the lateral wall, and thus is fixed in position; otherwise its thinness of structure would cause it to be liable, if subjected to undue muscular stress or to the pulling of inflammatory scar tissue, to sag towards the outer wall. With the tonsil removed the palatopharyngeus inevitably becomes endangered from any force, whether muscular or contraction of scar tissue, pulling the muscle in either direction. Should this lateral displacement result in close adhesions to the outer wall, not only might the muscle be impaired in its action or even be entirely prevented from functionating, but more important still, the palate might be so closely held down as to prevent its upward movement on phonation, in which case a permanent nasalized voice, due to operative rhinolalia aperta, would inevitably result.

The second purpose served by the tonsil to the palatopharyngeus is protective. This protection is most important at two points. The first of these is at the angle formed by the palatoglossus and palatopharyngeus as they emerge from the palate. Here the tonsil with its mucous membrane covering insinuates itself into the depths of this angle, thus preventing adhesion between the two muscles. Tonsillectomy by removing this protection exposes the muscles to the danger mentioned. The second point at which the protection of the muscle is especially important to the palatopharyngeus is the point of passage of the muscle into the pharyngeal wall. Here pharyngeal surface and muscle necessarily are in close association. If the tonsil were absent, either traumatic or inflammatory lesions would be much more likely to produce impairing injury to the muscle. In the operation of tonsillectomy, to remove the tonsil from the palatopharyngeus to this low point involves danger of adhesion, from injury to the tissues (for the capsule is here quite closely adherent) and from the inevitable scar tissue even without such injury. The danger is emphasized when one notes that the same phenomenon of adhesion occurs at this point in a mild degree not uncommonly through inflammatory processes, even in nontonsillectomized throats. In so far as the layer of mucous membrane which curves from behind the posterior pillar around its free border on to the anterior surface of the pillar and so on to the surface of the tonsil is well preserved, will it help to perform the protective work of the tonsil; and its preservation, as has been remarked by others, is important.

Summarizing now the function of the tonsil with reference to the action of the depressor palatal muscles, as indicated anatomically, we find with reference to the palatoglossus muscle:

1. The tonsil plays with contiguous structures a necessary part in the creation of a channel for the muscle which determines its location, length, direction of action, and protection against injury through movement of adjacent muscles. If the tonsil be extirpated the muscle is left suspended beneath a thin ledge of mucous membrane, which, being unsupported at its inner border, is liable to the following fates: (1) Slight or moderate contraction and retraction of the ledge in both a lengthwise and a lateral direction; (2) marked retraction in a lateral direction, possibly without adhesion; although adhesion is very likely to occur, in which case there results (3) marked retraction with partial adhesion, either (a) anteriorly, or (b) posteriorly; or (4) adhesion to the lateral wall throughout its linear course.

With reference to the palatopharyngeus the tonsil plays a part less vital, to be sure, than that played for the palatoglossus, but nevertheless a part of great importance. Especially is this true in view of the larger size of the muscle and its important relation to the larynx. With the palatoglossus extirpation of the tonsil must result, at any rate theoretically, unless in cases of exceptional anatomy, in impairment of the action of the muscle, which usually must be serious in degree, and must often reach total disability. On the other hand (not considering injury due to scar tissue at the surface of the muscle, the seriousness of which we are unable at this time to estimate), serious impairment to the action of the palatopharyngeus following tonsillectomy, would result only through direct operative injury to its fibers, or through the dragging of the weakened muscle out of place by the pulling of an adherent anterior pillar, or by the pulling of scar tissue, or by being tied down by adhesions, or by any two, or by all of these factors combined. (Figure XI, A.) Extirpation of the tonsil might result (1) in the sagging of the palatopharyngeus towards the outer wall; (2) in moderate adhesions at its point of entrance into the pharyngeal wall; (3) in marked adhesion to the lateral wall, thus largely incapacitating the muscle; (4) in the adhesions so dragging on the soft palate as to prevent its being raised against the pharyngeal wall for phonatory purposes.

STUDY OF TONSILLECTOMIZED THROATS.

We have carefully observed the tonsillectomized results in forty-three persons. The ages run from two to forty years; the operators number more than a dozen, most of them thoroughly experienced. We have tabulated the results according to the findings for each of the eighty-three tonsils operated. One factor which in tonsillectomy would inevitably tend to produce impairment in the action of the muscles concerned, that of the scar tissue formed on the bared surface of muscles, could not be estimated without microscopic study of tonsillectomized throats, and consequently has not been taken into consideration. The tabulation of the end results of tonsillectomy is as follows:

1. Neither the palatoglossus nor palatopharyngeus muscle shows more than slight retraction—i. e., both muscles appear essentially normal on both sides of the throat; three individuals; seven per cent.

2. Moderate retraction of the mucous membrane ledge (palatoglossus) in a lateral direction without evident adhesion; partial impairment of action of muscle; fifteen tonsils; eighteen per cent.

3. Marked retraction of the mucous membrane ledge (palatoglossus) in a lateral direction, but without evident adhesion; considerable impairment of muscle; seventeen tonsils; twenty-one per cent. (Figure VII.)

4. Marked retraction of the mucous membrane ledge (palatoglossus) in a lateral direction, with adhesions (a) anteriorly; decided impairment of muscle; five tonsils; six per cent (Figure VII); and (b) posteriorly; total incapacitating of muscle; thirteen tonsils; fifteen per cent. (Figure VIII.)

5. Adhesion of the mucous membrane ledge (palatoglossus) throughout its entire linear extent; total incapacitating of

muscle; twenty-four tonsils; twenty-nine per cent. (Figure IX.)

6. Palatopharyngeus appears normal or nearly normal; sixty-three tonsils; seventy-four per cent.

7. Slight or moderate pulling of palatopharyngeus towards lateral wall with adhesion; partial impairment of muscle; eleven tonsils; thirteen per cent. (Figure XI, B.)

8. Marked adhesion of the palatopharyngeus to the lateral wall; decided and possibly total incapacitation of the muscle; six tonsils; seven per cent. (Figure XI, A.)

It seems conservative to say that following tonsillectomy the palatoglossus and palatopharyngeus muscles are never wholly normal in their action. A striking fact concerning the appearance of the pillars in nontonsillectomized throats is a certain ease and freedom of movement, due to the provision of an abundant amount of tissue, allowing for full, easy range of action. This picture in tonsillectomized throats is altered for one of deficiency of tissue and tenseness of action.

The agreement of the findings above of the results of tonsillectomy, with the anatomic indications of the function of the tonsil with reference to the depressor palatal muscles, need hardly be pointed out. Anatomically the tonsil seems vital to the action of the palatoglossus; and in the tonsillectomized throats, the muscle is found to be to the eye practically normal in only eighteen per cent, impaired in forty-five per cent, and totally incapacitated in thirty-nine per cent. Anatomically the tonsil appears important to the perfection of action, though not vital to the function, of the palatopharyngeus, and in the tonsillectomized results it is found normal to the eye in seventy-four per cent, and impaired in nineteen per cent.

It has seemed to us best in this article to confine ourselves to the one question of the part played by the tonsil in the action of the depressor palatal muscles. The all important question of the danger of tonsillectomy to the voice, whether the speaking or the singing voice, necessarily is closely involved in the matter under discussion. The subject of the voice in this connection is, however, far from simple, and too important to be casually dealt with. It goes without saying that one who undertakes an operation necessarily so hazardous to the action of the depressor palatal muscles, as indicated by the findings just reported, is playing with fire so far as the voice, and especially the singing voice, is concerned. We feel that it might create a wrong impression, however, if we did not at any rate tabulate the voice cases included in the above list of tonsillectomized throats.*

Speaking voice apparently unimpaired: thirty-eight individuals, eighty-eight per cent; speaking voice showing rhinolalia aperta (open nasaling), due apparently to marked tension exerted on the soft palate by close adhesions of the palatopharyngeus to the lateral wall: three individuals; seven per cent; speaking voice impaired, but apparently not due to the operation: two individuals, five per cent; singing voice impaired (as to high pitched notes only): one individual, two and five-tenths per cent. It should be added that in two individuals (five per cent) a "drawing sensation" in the tonsil region was present; the sensation in one case, a young woman of twenty-two years with much scar tissue, was highly annoying.

SUMMARY AND CONCLUSIONS.

1. The tonsil serves as an absolutely necessary factor in providing a channel for the action of the palatoglossus muscle.

2. The function of the tonsil with reference to the palatopharyngeus is to afford support and protection of great importance to its normality of action.

3. Tonsillectomy serves to destroy not merely a possible lymphatic function of the tonsil, but also to either disturb or destroy an important physicomechanic function, one which is capable of being clearly understood.

4. More or less impairment of action of the depressor palatal muscles must occur in practically all cases following tonsillectomy, regardless of the delicacy of operative technic or the particular form of operative procedure adopted; but delicacy of procedure and method of operation are not, of course, to be considered unimportant.

5. To consider the present operation of tonsillectomy as a final settlement of the operative approach to the tonsil is pre-

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*We have left out of consideration entirely one case of badly injured palate with marked impairment of the speaking voice; the case is highly suggestive, however, of the dangers to the voice from so difficult and radical an operation in the hands of the untrained.

mature and erroneous. The whole tonsil question requires further anatomic, pathologic and operative study, in order, if possible, to readjust the operative approach to the organ to the new knowledge which is accumulating.

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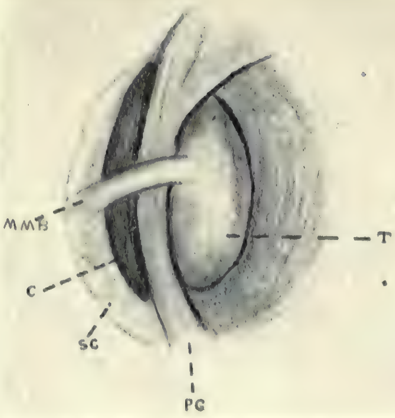

Fig. I.

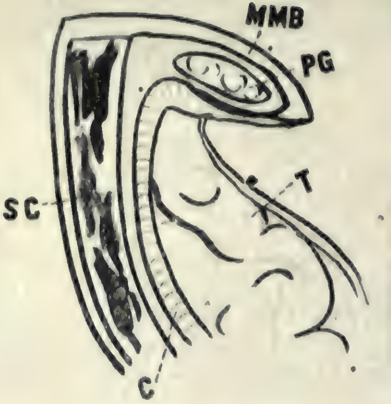

Fig. II A.

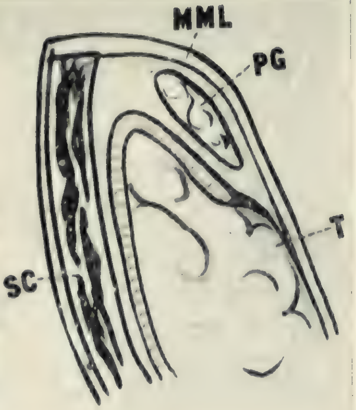

Fig. II B.

Fig. I. Drawing from dissection, showing relations of palatoglossus muscle. P. G., palatoglossus; T, tonsil; C, anterosuperior border of capsule of tonsil; SC., superior constrictor muscle; MMB, a section of the "mucous membrane bridge," which elsewhere in the drawing has been removed.

Figure II, A and B. Showing diagrammatically in cross section the structures and relations which determine the action of the palatoglossus. In A the amount of tonsillar lymphatic tissue is small, in B large; T, tonsil; PG, palatoglossus muscle; MMB, "mucous membrane bridge"; SC, superior constrictor muscle; C, anterosuperior border of capsule of tonsil.

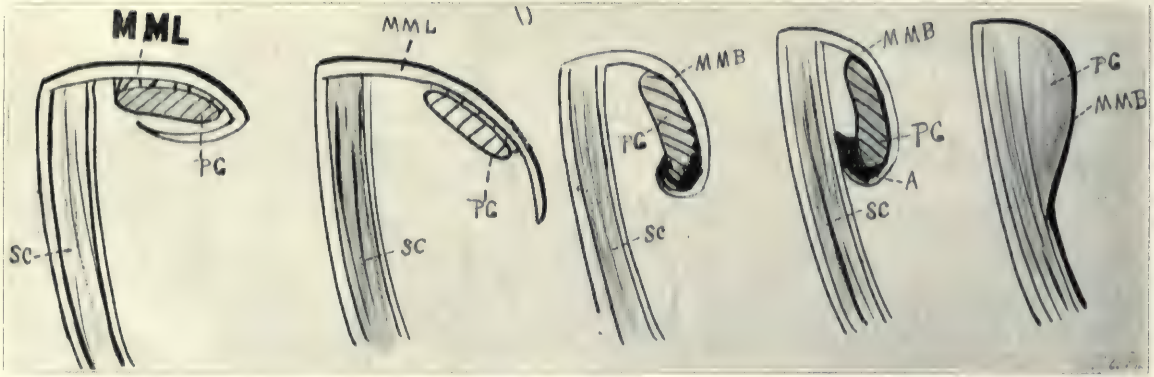

Fig. III A. Fig. III B Fig. IV A. Fig. IV B. Fig. IV C

Figure III, A and B. Showing the predicament of the palatoglossus muscle after the tonsil has been extirpated. These figures correspond respectively to A and B of Figure II. PG, palatoglossus; MML, "mucous membrane ledge,"; SC, superior constrictor muscle.

Figure IV, A, B, C. Showing the possible final fates of the palatoglossus following total extirpation of the tonsil. A, retraction of "mucous membrane ledge" with the palatoglossus; B, palatoglossus adherent to outer wall; C, identity of PG and "mucous membrane ledge" completely lost in a mass of adherent scar tissue; PG, palatoglossus; SC, superior constrictor; MMB, "mucous membrane ledge."

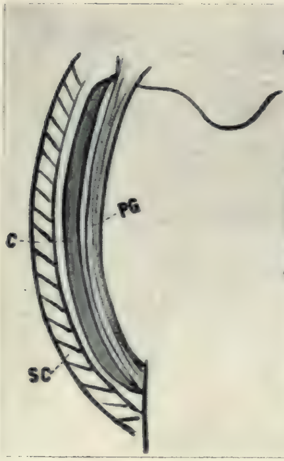

Fig. V.

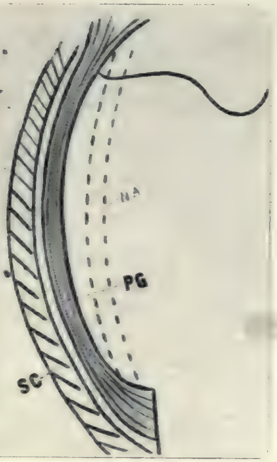

Fig. VI.

Figure V. Showing normal direction of action of palatoglossus muscle, and normal relation of anterosuperior border of the tonsillar capsule to the palatoglossus and superior constrictor muscles. PG, palatoglossus; C, anterosuperior border of tonsillar capsule; SC, superior constrictor.

Figure VI. Showing position of palatoglossus muscle following total extirpation of tonsil, due to retraction of "mucous membrane ledge"; adhesions of palatoglossus to outer wall absent. NA, normal direction of action of palatoglossus; PG, palatoglossus; SC, superior constrictor; A, adhesion.

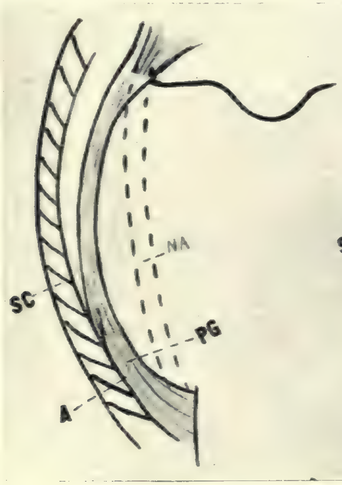

Fig. VII.

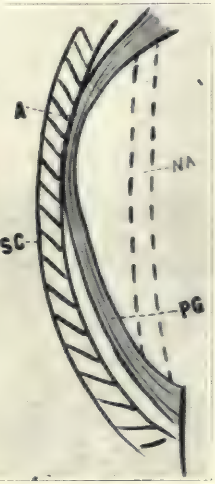

Fig. VIII.

Figure VII. Showing adhesion of palatoglossus to outer wall following total extirpation of tonsil, in anterior portion only. (For lettering see Figure VI.)

Figure VIII. Showing adhesion of palatoglossus to outer wall following total extirpation of tonsil; adhesion at palatal end only. (For lettering see Figure VI.)

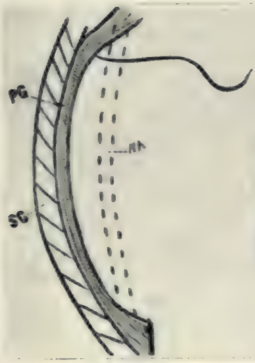

Fig. IX.

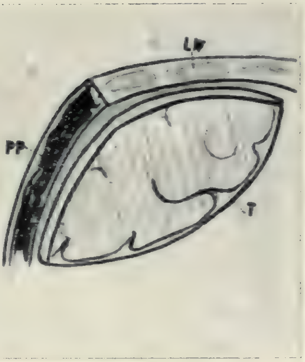

Fig. X.

Figure IX. Showing complete adhesion of palatoglossus to outer wall following extirpation of tonsil. (For lettering see Figure VI.)

Figure X. Showing the manner in which the tonsil acts as a supporting brace to the palatopharyngeus muscle. LW, lateral wall; PP, palatopharyngeus muscle; T, tonsil.

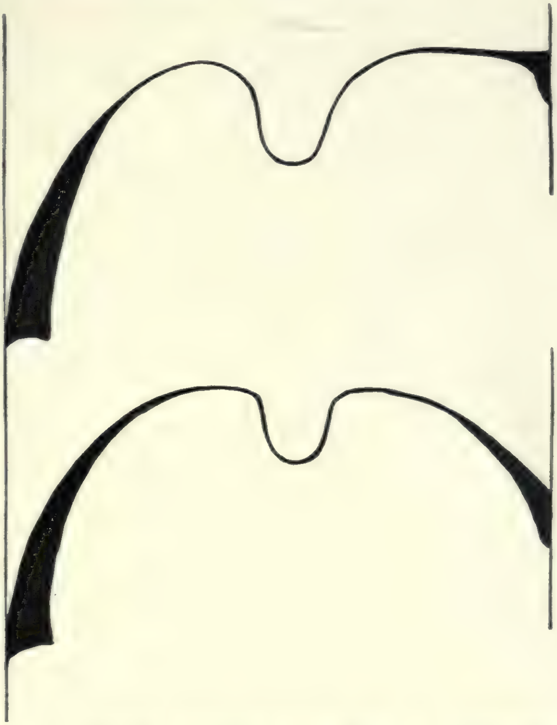

Figure XI. Diagram showing the result of adhesions of the palatopharyngeus to the outer wall. A, marked; such cases are liable to permanent impairment of the speaking voice. B, moderate; might occur from too low dissection of capsule.

L.

INVOLVEMENT OF THE LABYRINTH BY WAY OF THE DUCTUS ENDOLYMPHATICUS

By Norval H. Pierce, M. D.,

Chicago.

Reports of cases of infection of the saccus secondary to labyrinthine suppuration are very numerous, the labyrinth, in such cases, being involved through a fistula of the horizontal semicircular canal, the fenestrum ovale, or the promontory. The inflammatory process, spreading to the ductus cochlearis, thence through the ductus endolymphaticus, invades the saccus, and there forms a larger or smaller accumulation of purulent material.

This has been thought to be one of the paths by which cerebellar abscess may originate, but most frequently the end result is a sub- or an intradural abscess, as the sac has a far greater tendency to burst toward the bone than toward the arachnoid space.

Involvement of the labyrinth secondary to inflammation of the saccus, or by way of the ductus endolymphaticus, is very difficult to prove postmortem; the literature on the subject is, therefore, very meager. Cases have been reported in which there has been found no demonstrable fistula in any of the semicircular canals, the footplate of the stapes, or the promontory, and in which the labyrinthine suppurative process has eventuated in a fatal meningitis, the saccule having been proved, microscopically, to have been involved directly from the bone upon which it lays, this having become softened in the course of an acute or chronic mastoiditis, or in the course of a sigmoid sinus thrombosis.

During the last five years I have had two cases which had about the same characteristics and which were as follows:

Acute mastoiditis, following an attack of acute otitis media suppurativa. After subsidence of the initial otitic symptoms in the course of a month, they displayed temperature, more or less constant; absence of marked external tenderness and infiltration, headache, corresponding to the side of the ear involved; decrease in bone conduction; a slight decrease in air conduction, giving a positive Rinne; marked lowering of the upper tone limit, with preservation of the lower tone limit; nystagmus, rotatory in character, to both sides—that is, to the sound and the diseased side—when the eyes turned in these directions. The static labyrinth responded to the caloric tests; there was no stiff neck, no Kernig, no Babinski; but the spinal fluid was somewhat turbid, the cell count being increased from two hundred to five hundred per centimeter.

In the course of a simple mastoid operation marked softening of the temporal bone upwards and backwards was found. In both cases the tegmen antri could be removed with a sharp spoon. As far as could be ascertained upon the table, there was no fistula leading to the superior or horizontal semicircular canals.

The cases followed the usual course of healing, without any further disturbance, the meningeal tension ceasing in the course of three or four days, the nystagmus disappearing at the end of a week.

In my own mind I had explained these symptoms of labyrinthine irritation by assuming that inflammation of the dura had spread down to the eighth nerve and had caused the irritative symptoms of the static apparatus.

A recent case, however, has thrown some light on the subject. While, as in the two cases previously mentioned, the third resulted in recovery, thus excluding autopsy proof, the strong presumptive evidence would seem to indicate that the saccus endolymphaticus may have been involved in a plastic inflammation, producing severe disturbance of the labyrinth and symptoms of beginning meningitis. I will briefly relate the history:

Mr. E. O. B., lawyer, thirty-six years of age, entered St. Luke's Hospital, January 21, 1916, on the service of Dr. Theobald. He was a thin, pale man and had suffered from a severe double otitis media which followed an attack of grippe beginning January 1st. This was probably the first trouble he had ever had with his ears. Paracentesis was performed on both ears two days after the onset of the trouble. He had had scarlet fever, the usual diseases of childhood, and chronic ethmoiditis. Family history negative.

There was no pain on deep pressure over the mastoid and no swelling; there was a purulent discharge, considerable in amount, from both ears. The blood count was 8400 whites, sixty-eight per cent polymorphonuclears. The temperature ranged from 100° to 102.6°. Urine, sugar and albumin absent; microscopic examination negative.

The ear was packed with gauze, and a fifty per cent alcohol dressing applied. The temperature chart is displayed herewith, and may be consulted by those who wish to see for themselves.

While the temperature improved, the discharge continued until February 1st, when the temperature began again to mount up. The patient then complained of headache; had sleepless nights; his general condition was less favorable than when he entered the hospital; his tongue was furred; he was constipated. There was a trace of albumin, but no casts. In spite of the absence of mastoid swelling and of tenderness and pain over the mastoid, I advised operation. Skiagraphs taken on February 2d disclosed nothing definite.

In pursuance of this advice, on February 4th I opened both mastoid processes, under gas. There was a small amount of pus in each mastoid, in the region of the tip cells. There was no fistula in either external semicircular canal, but on the right side I found an area of softened bone, which could be easily removed with the curette, extending inward and backward around the region of the labyrinth, to a point above the upper border of the sigmoid sinus. The meninges were uncovered to the extent of about one-half centimeter. The dura looked hyperemic and slightly thickened.

The patient did not do very well after the operation. The various laboratory examinations will be found elsewhere. If you will consult the temperature curve, you will find that the temperature did not return to normal, but continued for some days to range from 100.5° to 99° and 98.8°. On February 13th the temperature was normal, the wound discharged freely, and there was a discharge from the external auditory canal. On February 17th the temperature began to mount to 100° and 100.5°.

He ran along thus until March 1st, when he complained of a sensation of dizziness and of being nauseated. The temperature on this day varied from 98.6° to 101°. The dizziness, nausea and vomiting increased during the day, so that he was unable to change position in bed, but remained on his left side. Every attempt to change position resulted in a violent attack of vomiting, acompanied by diaphoresis.

When I saw him the next day he had a violent nystagmus of the third degree to the left. The right static apparatus did not respond to caloric tests, and he was unable to hear a Bezold small a[1] fork when the noise apparatus was held in the left ear. He complained a good deal of deafness, headache, and indefinite stiffness about the neck. There was no Kernig and no Babinski, but there was marked photophobia. Pus from the ear showed hemolytic streptococcus; staphylococcus albus. On March 6th, spinal fluid showed Nonne strongly positive; Lange positive; cell count 95 per centimeter; culture negative; no sugar. On March 8th, Nonne strongly positive; Ross Jones strongly positive; Lange positive; cell count per centimeter, 973; polymorphonuclears, ninety-five per cent; cultures negative; sugar negative. The blood count throughout is given below.

EXAMINATION OF BLOOD.

Quantitative.

January 21, 1916—Leucocytes per c.m.	8,400

Differential Count.

Lymphocytes:	
(a) Small	23
(b) Large	4
(c) Indentate nucleus	3
Neutrophiles:	
(a) Polymorphonuclear	68
Eosinophiles:	
(a) Polymorphonuclear	1
Basophiles	1

Quantitative.

January 22, 1916—Leucocytes per c.m.	11,700

Differential Count.

Lymphocytes:	
(a) Small	16
(b) Large	6
(c) Indentate nucleus	1
Neutrophiles:	
(a) Polymorphonuclear	75
Basophiles	2

Quantitative.

January 23, 1916—Leucocytes per c.m.	8,300

Differential Count.

Lymphocytes:	
(a) Small	23
(b) Large	2
(c) Indentate nucleus	2
Neutrophiles:	
(a) Polymorphonuclear	73

Quantitative.

January 24, 1916—Leucocytes per c.m.	8,600

Differential Count.

Lymphocytes:	
(a) Small	20
(b) Large	6
Neutrophiles:	
(a) Polymorphonuclear	70
Eosinophiles:	
(a) Polymorphonuclear	2

Widal negative.

Quantitative.

January 25, 1916—Leucocytes per c.m.	7,000

Differential Count.

Lymphocytes:	
(a) Small	24
(b) Large	3
(c) Indentate nucleus	1
Neutrophiles:	
(a) Polymorphonuclear	71
Eosinophiles:	
(a) Polymorphonuclear	1

Quantitative.

January 27, 1916—Leucocytes per c.m. 7,200

Differential Count.

Lymphocytes:	
(a) Small	27
(b) Large	6
Neutrophiles:	
Polymorphonuclears	65
Eosinophiles:	
Polymorphonuclears	2

Quantitative.

January 31, 1916—Leucocytes per c.m. 8,000

Differential Count.

Lymphocytes:	
(a) Small	25
(b) Large	7
Neutrophiles:	
Polymorphonuclears	67
Basophiles	1

Quantitative.

February 17, 1916—Erythrocytes per cm.	3,800,000	
Leucocytes per c.m.	6,400	
Hemoglobin (method)	63	Sahli

Differential Count.

Lymphocytes:	
(a) Small	26
(b) Large	2
(c) Indentate nucleus	2

Neutrophiles:	
Polymorphonuclears	68
Eosinophiles:	
Polymorphonuclears	2

Quantitative.

February 24, 1916—Erythrocytes per c.m.	4,290,000	
Leucocytes per c.m.	9,000	
Hemoglobin (method)	68	Sahli

Differential Count.

Lymphocytes:	
(a) Small	24
(b) Large	4
(c) Indentate nucleus	1
Neutrophiles:	
Polymorphonuclears	70
Basophiles	1

Quantitative.

March 1, 1916—Leucocytes per c.m.	9,000	
March 2, 1916—Erythrocytes per c.m.	3,880,000	
Leucocytes per c.m.	10,200	
Hemoglobin (method)	64	Sahli

Differential Count.

Lymphocytes:	
(a) Small	20
Neutrophiles:	
Polymorphonuclears	80

Quantitative.

March 5, 1916—Leucocytes per c.m.	10,400

Differential Count.

Lymphocytes:	
(a) Small	16
(b) Large	3
(c) Indentate nucleus	2

Neutrophiles:	
Polymorphonuclears	78
Eosinophiles:	
Polymorphonuclears	1

Quantitative.

March 11, 1916—Erythrocytes per c.m.	4,080,000
Leucocytes per c.m.	6,700
Hemoglobin (method)	66 Sahli

Quantitative.

March 22, 1916—Leucocytes per c.m.	9,200

In the left ear he complained of great tinnitus. The audition in this ear was also very much impaired, but it responded to caloric stimulation. The trouble was evidently in the right labyrinth, and the laboratory findings indicate a beginning meningitis. At this time the labyrinth operation was very seriously considered. The temperature, March 6th, was 102.6°. However, on the following day his general condition seemed to be a good deal improved, the pain in the head had subsided, the temperature was lower, and the dizziness and vomiting were markedly less.

From this time on, with the exception of a few days, when there were occasional pains in the left ear necessitating the opening of the drum head, he continued to improve. He was discharged March 27th. He is now performing his duties. An examination, just before I left home, showed that he could hear the small a^1 forks, could distinguish spoken numerals about two feet from the right ear, with the noise apparatus in the left ear, demonstrating that function was returning.

We have here, therefore, a serous labyrinthitis resulting in a serous meningitis, occurring in an acute otitis media twenty-seven days after the mastoid operation, and fifty-two days after the onset of the ear rtouble.

What of the presumptive evidence that involvement of the labyrinth occurred by way of the ductus endolymphaticus? The labyrinth is involved scarcely ever through the semicircular canals in acute otitis media. The exceptions are cases of acute mastoiditis occurring in the course of severe scarlet fever, and in cases where there is a dehiscence of the external semicircular canal. As we had the opportunity to inspect the external semicircular canal, we may at least exclude this canal from macroscopic changes. This may be said also regarding the posterior and superior canals, which may also be excluded as gateways of infection, as there was healthy bone lying between them and the softened fistula which led to the posterior surface of the temporal bone. It is hardly possible that it could have occurred by way of the footplate of the stapes, or the fenestra ovalis, as the ear was drained thoroughly both by paracentesis through the tympanic membrane and posteriorly into the mastoid wound. The same may be said of the promontory.

If there had been a dehiscence of the external semicircular canal, which is very rare in adult life, or a dehiscence in the promontory, which is still more rare, the symptoms of involvement of the labyrinth would have occurred much earlier in the course of the disease.

The softened bone which I mentioned as having been present at the time of operation in the right ear, which was later involved in the labyrinthine trouble, extended directly to the meninges, which were very close to the situation of the aqueductus vestibuli. This I ascertained by boring several healthy bones in the direction taken by the fistula, finding that they very closely approximated the neighborhood of the saccus endolymphaticus.

In conclusion, I would emphasize a few points:

1. That the saccus endolymphaticus may be involved more frequently than is usually supposed in acute softening processes of the mastoid.

2. That it is our duty in all cases of mastoid involvement without external manifestations except temperature, to very thoroughly search for softened areas deep down in the bone, and not cease until we come to healthy structure.

3. That we should always bear in mind in suppurative labyrinthitis not only to open into the bony labyrinth, but to explore the region in which the saccus endolymphaticus is situated.

4. That within the first few days serous labyrinthitis cannot

be differentiated from suppurative labyrinthitis by any diagnostic means at present at hand.

5. That absence of globulin, presence of lactic acid, absence of sugar, may be noted in inoffensive serous meningitis.

6. That in these three cases viable microorganisms were not recovered from the cerebrospinal fluid, which, in my opinion, is the only differentiating point between spreading suppurative meningitis and serous or circumscribed meningitis.

7. Several lumbar punctures were made in the course of the week following the appearance of meningeal symptoms; that is, whenever the headache, stiffness of the neck and temperature increased. These punctures were invariably followed by improvement, as evidenced by decrease in headache, stiffness of neck, and temperature.

LI.

REPORT OF A CASE OF SYRINGOMYELIA WITH MARKED VESTIBULAR SYMPTOMS.*

By G. E. Shambaugh, M. D.,

Chicago.

An important chapter has been added to otology by the recent studies in the physiology of the vestibular part of the inner ear and by the analysis of the symptoms arising from disease of this mechanism. The pursuit of these studies has led the otologist into an examination of the symptoms arising from disturbance of the central tract of the vestibular impulses, and has thus brought his work into close relation with that of the neurologist. In making a diagnosis of intracranial lesions the neurologist can often be assisted by the intelligent cooperation of the otologist.

In our studies of the vestibular mechanism we have already arrived at a satisfactory working basis for the interpretation of the symptoms arising from disease of the labyrinth and for the diagnosis of the several forms of labyrinth disease. On the other hand, in the interpretation of the vestibular symptoms arising from disease of the central nervous system, and in the diagnosis of these central lesions, we have made scarcely more than a beginning. We are still apparently on the threshold of what is to be a most valuable field for work. For these reasons it is still of great importance that cases should be reported where a diagnosis of disease of the central nervous system can be established, and in which there are distinct vestibular symptoms. These considerations have led me to report here the following case of syringomyelia producing symptoms arising from disturbance of the vestibular tract.

The case is that of a young man, aged twenty-eight years, a mining engineer, who was referred to me in September, 1915, because of a disturbance of equilibrium and sensations of vertigo. He dates the onset of his difficulty two years ago, since when he has suffered most of the time from headache, chiefly occipital. The disturbance of equilibrium consists of a sense of instability when walking, associated with some sensation of vertigo and occasional slight nausea. Ever since the onset of this trouble he has experienced much difficulty in reading. He discovered that in order to read at all it became necessary for him to hold the paper far over to one side. At the same time he would turn his face in the opposite direction. Only in this way, by making as strong a deviation of the eyes as possible, was he able to read. The side toward which the eyes had to be directed was not always the same, sometimes being to the right, at other times to the left. This change of sides often came on at short intervals. He would discover while reading with the book held over to one side, that the page became blurred, and that by holding the book over to the opposite side he would again be able to read. At no time had he noticed any tinnitus aurium or any difficulty in hearing.

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*Read before the American Otological Society, Washington, D. C., May, 1916.

In making an examination of his ears the drum membranes were practically normal, and the functional examination disclosed normal hearing in both ears. There was spontaneous nystagmus present on looking straight ahead. The eye movements were rather coarse and chiefly in the horizontal plane. The direction of the nystagmus was now toward one side, now toward the other, with but a few seconds' interval, during which the eyes would remain quiet. On deviation of the eyes toward the side of the quick component the nystagmus became much more marked. Deviation of the eyes toward the slow component changed the character of the nystagmus. The movements became less marked, and the quick component was directed toward the side of the deviation. It was found that his difficulty in reading was due to the violent spontaneous nystagmus, and the deviation of the eyes, now to one side, now to another, while reading, was an effort to reduce to its minimum the ever-present spontaneous nystagmus. For example, it was noticed that when the spontaneous nystagmus was directed toward the right, he would hold the book over to the left.

On making the rotation tests, it was noticed that when rotated toward the side of the slow component of the spontaneous nystagmus, this nystagmus was very much increased, and this increase seemed to last about a minute and a half before subsiding. When rotated toward the side of the quick component, the resulting after-nystagmus was much less violent, and was directed to the opposite side. This after-nystagmus would usually continue about one minute before it would cease, and there would be a return of the preexisting spontaneous nystagmus toward the opposite side.

Caloric stimulation, cold water, head erect, produced in either ear a ready response. The resulting nystagmus, while directed always toward the opposite side, differed from the normal reaction in that the character of the nystagmus was chiefly horizontal and only very slightly rotary.

The pointing tests also gave abnormal responses. The spontaneous pointing, shoulder from above, for both the right and the left hand, was normal, irrespective of the existing spontaneous nystagmus. After caloric stimulation (cold) of the right ear, pointing for the right hand, shoulder from above, produced a deviation to the right of only four to five inches, while for the left hand there was no deviation at all. Caloric stimulation of the left ear (cold), shoulder from above, both right and left hand touches. Here, in spite of the marked nystagmus to the right, no past pointing to the left was produced.

Pointing tests after rotation also failed to give the normal past pointing. After rotation to the right, pointing shoulder from above, the right hand showed past pointing to the right of only four inches, while for the left hand there was no past pointing. After rotation to the left, no past pointing for either hand could be produced.

The absence of any hearing defect in this case and the fact that both rotation and caloric stimulation resulted in a prompt response from the vestibular apparatus, showed clearly that the vestibular symptoms did not arise from disturbance in the internal ear, but was due rather to some intracranial lesion. The shifting of the spontaneous nystagmus toward either side was also clearly not the result of a peripheral disturbance, but could only be accounted for on the basis of some central disease. The failure to get the normal past pointing after stimulation of the peripheral mechanism, by either caloric stimulation or after rotation, was also clearly the result of some disturbance of the central tract of the vestibular impulses. In a case where there is no disease of the central tract, any disturbance of the peripheral mechanism which results in a disturbance of equilibrium is likely to produce spontaneous past pointing. This spontaneous past pointing will be toward the side to which the disturbance of equilibrium inclines the patient to fall. The past pointing will, in other words, be toward the side to which the slow component of the existing nystagmus is directed. For example, in a case where the disturbance of equilibrium is caused by the sudden suppression of function in one labyrinth, the tonus impulses from the opposite side, acting without the restraint of the normal tonus from the destroyed ear, will tend to throw the patient toward the affected side, and the slow component of the existing nystagmus will also draw the eyes toward the affected side. In the same way, while making the pointing tests in such a case, the arm will obey the same impulse, affecting all the other skeletal muscles, and there will be a spontaneous past pointing toward the affected side. Again, when in a normal case we produce a disturbance of equilibrium by disturbing the normal flow of impulses from the two ears, whether by means of rotation or by caloric stimulation, the induced past pointing will be toward the side to which the patient is inclined to fall—that is, toward the side to which the slow component of the nystagmus is directed.

When we come to the cases of disturbed equilibrium resulting from diseases of the central tract of the vestibular nerve, the past pointing often fails to conform to the above simple rules. In the case here reported, for example, the existence of a strong spontaneous nystagmus failed to produce spontaneous past pointing as it would probably have done had the nystagmus been due to a disease in the peripheral mechanism. Moreover, the stimulation of the peripheral mechanism by rotation and by cold water, while it produced a marked nystagmus, did not bring out the typical past pointing.

When it became clear that the disturbance in equilibrium in this case was the result of the disease in the central nervous system and not of the peripheral mechanism of the labyrinth, I requested Dr. Thor. Rothstein, a neurologist, to make an examination and determine the probable nature of the lesion. Dr. Rothstein discovered the case to be one of syringomyelia, with the extension of the lesion upward into the region of the fourth ventricle. The report of this examination is here appended:

DR. ROTHSTEIN'S REPORT.

"The patient states that he has enjoyed good health up to two years ago, when his present trouble started to develop. Many years ago he had observed that he could stand without discomfort much hotter and colder baths than other people. Six years ago he noticed that pinching of the skin did not cause pain over an area larger than the size of his hand and located on the left side, at the level of the heart region. Up to two years ago he was able to perform his work without any trouble, but at this time he started to become tired doing his work. He had to climb ladders in the mine, and he has found this tired him more and more during the last two years, and his mother states that she noticed a marked change in him during the last two years. He is more easily tired and does not laugh as heartily as he used to. Gradually some disturbance in walking has developed. He cannot handle his legs as easily as he used to and he has some vertigo. Also it has become difficult for him to read. (See description of nystagmus.) He suffers at times from headaches, but they are never severe, and he also has some pain in the back when tired.

"Physical Examination.—He is a well developed man and muscular. No paralysis of any muscles of the body exists. No atrophy of the muscles. Although he can walk, as it seems, unhindered, the legs are slightly dragged and are kept adducted, but his steps are of about the normal length. With the exception of the nystagmus, nothing abnormal is found about the eyes, pupils reacting to light and accommodation and the eye grounds being normal. Triceps, radial and ulnar reflexes lively and present on both sides. Patellar reflexes increased. Ankle clonus slight on both sides. Plantar reflexes present. Babinski reflex at times on both sides. Cremasteric reflex present. Abdominal reflexes lost on left side but present on the right. There is a slight resistance to passive movement of the legs, but this is not marked.

SENSATION.

"Sensation to Touch.—Right side of body: Sensation to touch is normal.

"Left side of body: Sensation to touch is considerably decreased, but not entirely lost, over an area extending between the level of the mammila and the level of the lower border of the thorax. It is less markedly decreased between this last level and the level of the crista ilii, and only slightly decreased over the area innervated by the second and third lumbar roots. On the rest of the left side of the body sensation to touch is normal. The decrease in sensation to touch is found both anteriorly and posteriorly.

"Sensation to Pain.—Right side of body: Sensation to pain is normal.

"Left side of body: Sensation to pain is lost entirely over an area between the level of the mammila to level of the lower border of thorax. It is markedly decreased over the area between the level of the lower border of the thorax and crista ilii and over the area of the second and third lumbar roots. It is also slightly decreased between the level of the mammila and the level of the clavicula. The decrease in sensation to pain is also found both anteriorly and posteriorly.

"Sensation to Temperature.—Right side of body: Twenty-four degrees Centigrade is felt cold all over right side, but sensation to warmth is markedly decreased over an area between the level of the clavicular and lower border of thorax, and also decreased over ulnar half of right arm and hand.

"Left side of body: Sensation to temperature is practically lost over whole left half of trunk, with the exception of above level of second rib. It is markedly decreased over the area of second and third lumbar roots and ulnar half of left arm. The sensation to temperature, furthermore, is not quite normal on the neck and back of head, but is normal over area innervated by trigeminus.

"Wassermann on blood and spinal fluid is negative. In spinal fluid were found seven cells to cubic millimeter. Nonne test negative and Lange test negative."

CONCLUSIONS.

It is evident that a long-standing disturbance of sensation to temperature (see history), the present marked disturbances of sensation to temperature and pain, and the much less marked disturbances of sensation to touch, make a diagnosis of syringomyelia the only one possible. The condition of the reflexes, as well as the slightly marked motor disturbances, conform well to this diagnosis. The history and the condition of the spinal fluid excludes syphilis. The decrease in sensation to temperature over the neck and back of head, demonstrates that this syringomyelic process has reached up to the medulla oblongata. It is therefore quite natural to assume that the nystagmus in this case depends upon an extension of the syringomyelic process up into the region of the fourth ventricle (syringobulbia). Nystagmus has been described in syringomyelia by many authors (Grund, Thomas, Monro, Köliechen, McLaren, Bárány, and others), and in some cases as the only present supramedullary symptom. In Bárány's cases (two) there existed strong spontaneous nystagmus, but without inhibition nystagmus.

LII.

MASSAGE OF THE EUSTACHIAN TUBE.*

By Alfred Lewy, M. D.,

Chicago.

Since the advent of the nasopharyngeal speculum and the nasopharyngoscope, considerable progress has been made in the treatment of diseases of the ear dependent upon lesions of the eustachian tube and its immediate neighborhood. But while the removal of growths, breaking up of adhesions, bougieing and topical applications have attracted the attention of the inventors of these instruments, and that of others, so far as I could find, only casual mention was made in medical literature of massage of the eustachian tube until the subject was taken up last year by P. J. Mink of Utrecht, Holland.[1] For some time the osteopaths have been practicing massage of the tube by the finger, and claiming excellent results in some forms of deafness, but I have been unable to follow their diagnoses as described in their journals.

Mink describes his technic in detail, and reports a number of cases so treated. He believes this treatment will prove a valuable addition to our therapeutic resources. After a limited experience I believe likewise, but realize, of course, that it is useful only in properly selected cases, as an addition to, but not a substitute for other well established procedures.

The technic is easily carried out by anyone familiar with the use of the eustachian catheter. A nasal applicator is curved to about the same degree as an eustachian catheter, but a shorter segment of the curve is used. A number of different lengths of curve are necessary to fit septal deviations and various anatomic conditions. Mink uses a wire looped back on itself in opposite directions at either end, and curved to fit individual cases. One end serves as a handle, the other as a cotton carrier. The cotton is best folded back on itself and wound around the carrier so as to form a thick ball rather than a brush, to avoid trauma. After cocainizing the region of the mouth of the tube (by the same motion as hereinafter described), the cotton-wound applicator is passed back to the posterior wall of the pharynx, swept upward in the fossa of Rosenmueller, brought forward over the mouth of the tube and immediately downward and back under the mouth of the tube. This motion is repeated about twenty times, or as often as the operator's judgment indicates. This movement Mink describes as his "Greater stroke" (Grosser strich). Another movement he calls the "Lip stroke" (Lippenstrich). For this stroke the applicator is rotated upward and backward into the mouth of the tube, as is done in passing the catheter directly in. This dilates the mouth and exercises the muscular and other attachments. Still another movement is the "Small stroke" (Kleinerstrich), which is reserved for the cases in which he is unable for any reason to circle the mouth of the tube. This stroke is up and down in front of the tube. The cotton should be in fairly firm contact with the tissues throughout the movements. He does not describe the use of medicaments in connection with the massage, but in the cases so treated I used argyrol twenty per cent, iodoglycerin one-half per cent and ichthyol ten to twenty per cent. The latter, while useful, is, of course, disagreeable in taste and smell, besides being more irritating in the nasopharynx than in the nose.

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*Read before the Chicago Layngological and Otological Society, April, 1916.

The cases which I shall report were not selected especially because they were appropriate for this treatment, but rather for the variety of conditions on which it was tried.

Six cases of acute suppurative otitis media in which massage with argyrol was begun when acute symptoms had subsided and they were in the declining or stationary stage. I believe these cases were shortened in their course and hearing restored more rapidly than with simple inflation and topical applications as heretofore used.

One case of acute suppurative otitis media, with visible, pinhead size perforation, anterior inferior, apparently became dry and then relapsed after three weeks of treatment, since which I have not seen the patient. This patient also had moderate crusting, not of the ozenic type, in both nasal cavities, the source of which I had not discovered up to the last treatment.

Three cases of simple tubal catarrh of moderate degree yielded to a very few treatments.

Several more complicated cases I shall report in greater detail.

Case 11.—The result in this case was perhaps the most striking of all of them. The patient, a physician, aged fifty-three years; deafness many years, due, I believe, to a nerve degeneration plus tubal inflammation. He suffered from tinnitus, an almost constant feeling of fullness, squeaking noises on blowing nose or swallowing, and autophonia of such degree that it sometimes interfered with his business. He had suffered much at many hands, including my own, and had been applicated, bougied, and the socalled adhesions in the fossa of Rosenmueller broken up, but with no permanent relief. Massage of the tube, first with argyrol, later with ichthyol, about ten treatments, have given him marked relief. There have been no treatments for about six weeks, and no return of symptoms in spite of frequent damp days, which always heretofore aggravated his symptoms. The tinnitus remains in lesser degree, and the hearing is improved about as much as one would expect from relief of the subjective disturbances. The hearing test follows:

Right		Left
2′	Whisper	16″
	Slight improvement after inflation	
	Schwabach (A) 40″ (about normal)	
	Weber (A) to right (better ear)	
+ 12/35	Rinne	+ 12/35
	(Air and bone conduction shortened)	
24 d.v.	Low Limit	24 d.v.
Bone, a_6; Air, E_6	High Limit	Bone, a_6; Air, f_6
	(Some loss by monochord, nearly normal by E_4)	
Little opaque and retracted	Membrana Tympani	Little opaque and retracted

Case 12.—Male, aged fifty-six years; locomotor ataxia, which has improved under intraspinal injections of salvarsan-

ized and mercurialized serum. Deafness; ringing noises, feeling of stoppage; postnasal discharge; sinusoidal deviation of the septum.

Right		Left
	Whisper	
4′		5′
After inflation, 10′		No improvement on inflation
	Schwabach (A) 18″, 50%	
	Weber (A) to right (better ear)	
	Rinne	
+ 12/40		+ 12/40
	(Air and bone conduction shortened)	
	Low Limit	
24 d.v.		24 d.v.
	High Limit	
Normal		Normal
	Membrana Tympani	
Normal		Normal

After two months' biweekly massage with iodoglycerin, hearing right ear, sixteen feet before inflation; left ear, only slight improvement. Postnasal discharge only slight and occasional (there was no special treatment for this). Ringing diminished.

Case 13.—Male, aged thirty-nine years; uses much tobacco, chewing and smoking; deafness one year, especially for telephone; buzzing tinnitus. Chronic constipation.

Right		Left
	Whisper	
5′		6″
	No improvement on inflation	
	(A) Schwabach	20″ (About 50%)
	Rinne	
+ 12/40		+ 12/40
	Bone and air conduction shortened	
	Low Limit	
24 d.v.		36 d.v.
	High Limit	
Normal for c_4		Normal for c_4
	Eustachian Tube	
Open		Open
	Membrana Tympani	
Normal		Normal

Two months later, whisper, right ear, twelve feet; left ear, eighteen inches. Iodoglycerin massage was used, but I believe the improvement resulted largely from withdrawal of the tobacco and use of strychnin. About three weeks after last report above, sudden increase in deafness in right ear (whisper reduced to two feet). I put the patient on sweats and potassium iodid, but have not yet retested him, nor have I solved the cause of the recent increase of deafness of right ear.

Since writing the foregoing, four weeks ago, this patient has returned. He took potassium iodid about a week, then of his own accord took cod liver oil. He has had no local treatment, but his hearing for the whisper in the right ear is now nine feet; left ear, one foot. He is again using tobacco freely.

Case 14.—Female, aged forty-six years; dressmaker. About a week before coming to me a ringing suddenly began in the left ear; cause unknown; family history negative; sore throat every winter; tonsils small, but with dilated crypts, two of which contain débris.

Right		Left
	Whisper	
8′		18″
After inflation, 22′		After inflation, 30″
	(A) Schwabach, normal	
	Rinne	
+ 16/50		+ 16/40
	Bone conduction about normal, air somewhat shortened	
	Low Limit	
24 d.v.		24 d.v.
	High Limit	
Normal for c_4		Normal for c_4
	Membrana Tympani	
Slightly atrophic		Slightly atrophic
	Eustachian Tube	
Normal		Normal; few rales

After a few weeks' treatment, hearing in right ear normal; left ear, three feet for whisper. The tinnitus showed no special improvement until I bougied the left tube, which caused considerable improvement, but it is not yet entirely well. One month after this test had an attack of vertigo, but with no increase of deafness or tinnitus. In this case and in Case 13, the tonsils were not healthy, and may have been the source of infection causing the trouble, as recently described by Dr. Shambaugh. However, I believe in Case 13 the trouble was largely due to tobacco, and that the attack while under treatment was due to a relapse of the habit plus some other toxemia, as there was no evidence of recrudescence of the throat inflammation. The rôle of intestinal and other toxemias in nerve deafness has not yet been solved. Both Cases 13 and 14 had chronic constipation.

It is likely that some of you have used massage of the tube by some other method. I have tried it by using the vibrator on the mandible and side of the throat, but not with such good results as by direct use of the applicator.

So far I have had no special results from this treatment in tinnitus that failed to yield to other treatment, but I feel encouraged a little in some cases. The treatment seems especially useful to remove the results of tubal inflammation in cases of not too long standing.

REFERENCE.

1. Zeitschrift für Ohrenheilkunde, Bd. 72, Heft 3; Bd. 73, Heft. 1.

LIII.

DOUBLE MASTOID OPERATION, WITH REPORT OF CASE AND EXHIBITION OF SKIAGRAPHS OF MASTOIDS.*

By Norman H. Goodenow, M. D.,

Everett, Wash.

This case report, with exhibition of skiagraphs and patient, on whom I did a double mastoid operation, a radical on right, simple on left, three weeks ago today, is of more than common interest, as it shows how closely the pathologic findings during the operation coincided with the readings of the skiagraph, and the value of X-ray in making a diagnosis and deciding as to the treatment to be carried out to bring about a cure in such cases.

The patient, a young man, twenty-one years of age, physically well developed, and in good health, consulted me March 19, 1916.

History. A chronic discharge of foul-smelling pus from right ear, following an attack of purulent otitis media six years ago. Subjective noises and earache at times. Two years ago was operated by a general surgeon, which did not result in a cure. Inspection of external canal showed it to be filled with thick, very foul-smelling pus; after forcibly washing out with a large syringe, drum membrane found to be thickened, and a linear perforation extending transversely across drum was seen. Eustachian tube inflated through catheter, after which the hearing was watch at six inches; whisper, two feet; fork, lateralized to this ear, and bone conduction over mastoid shortened.

Left Ear. Six weeks ago began to notice subjective noises and impaired hearing in left ear. Consulted a Portland physician, who washed out some cerumen. Since that date there has been a free discharge of pus from this ear, but without pain and the usual symptoms accompanying an acute purulent otitis media. Inspection of this canal showed it to be filled with a thick pus, but without odor. This was syringed out, when a small polyp could be seen protruding through perforation in drum, which I removed with small forceps. Membrana tympani was thickened, moist and edematous. Inflation through catheter forced a mass of thick pus through perforation; this was removed, after which he heard watch at ten inches, whisper at three feet, bone conduction shortened.

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*Presented before the Puget Sound Academy of Ophthalmology and Otolaryngology, Seattle, Washington, April 24, 1916.

Examination of nose showed both sides free and without signs of pus. Throat revealed very large infected tonsils, the removal of which I advised as the first procedure to carry out in view of curing the chronic ear suppuration. This was done the next day under local anesthesia. During the week following both ears were treated by forcibly syringing out with large syringe, drying out carefully, powdered boric acid dusted in canals and a small strip of gauze inserted to facilitate drainage. Under this treatment the discharge and odor from right ear greatly lessened; the left drum, however, remained moist and edematous. Through the perforation in right drum a white mass could be seen, which proved to be cholesteatoma.

On March 28th a skiagraph was taken by Dr. J. Howard Snively. His interpretation of the plates was as follows:

"On the right side there has been a chronic mastoiditis. At the site of the operation the bone is very thin, and cells have been destroyed; surrounding this area is an area of dense ivory-like-bone, due to the chronic irritation and suppuration. Most of the remaining cells have been nearly obliterated by this sclerotic process. On the left side the cellular structure is discernible over the usual area, but the cells are clouded as though filled with inflammatory exudate rather than air. The condition here is more or less acute undoubtedly, as no breaking down or necrosis of the cellular structure is seen."

On March 31st the hearing of right ear was watch at four inches, whisper at about two feet, both air conduction and bone conduction shortened. Left ear, watch at ten inches, whisper at three feet, air conduction and bone conduction shortened. I now advised a radical operation to be done on the right mastoid and possibly same on left. Operated on April 3rd, just three weeks ago today.

Operation: In doing the radical on right mastoid, I made a curved incision, the convexity of which I carried well back of ear. This gives a large wide flap of skin and underlying periosteum to be used in covering the field of exenteration in the bone when the wound is sutured and closed, thus leaving the line of suture over the outer table in place of over the cavity, which I think is quite an advantage in the healing process. After exposing the mastoid process I found a dehiscence of the outer table just anterior to the lateral sinus, this being the site of the previous operation, filling in with new bone never having taken place. This gap in the bone was filled with granulations and a probe was easily passed forward directly into antrum. After clearing away the granulations and removing the bone covering the antrum, the lateral sinus was exposed for about an inch. The membrane covering the sinus was healthy, and a pad of gauze was laid over it to protect it from injury while completing the operation.

I now removed the posterior bony wall of the external canal down to a level with its floor, then, with a small sharp chisel and curette, the facial bridge was cut through and removed.

The antrum was found to be full of granulations and pus, a mass of cholesteatoma was removed from the tympanum. The remnants of the ossicles and drum was also removed with curette and small forceps; the eustachian tube curetted thoroughly. The posterior superior wall was also removed, so that there was left no overhanging wall. I then made two parallel incisions through the posterior membranous canal, making a tongue flap which was pushed back in the cavity, a packing of iodoform gauze inserted through the external canal and the primary incision closed up its whole length.

An interval of fifteen minutes was now taken to prepare for the operation on left mastoid, instruments were washed and sterilized, gowns and gloves changed.

On the left mastoid my findings pathologically were in accord with Dr. Snively's readings of the skiagraph. There was no necrosed bone, but the antrum was filled with granulations and pus. I was able to pass a good sized probe through aditus into the middle ear, thus being assured of good drainage. I did not think it necessary to do a radical on that side.

The patient made a rapid recovery; did not have a postoperative temperature; left the hospital the day of the first dressing, which was on the fourth day. The sutures were removed the next day and the wound of the radical found firmly united. The packing of the cavity of the left mastoid was discontinued and the wound closed at the end of two weeks.

There is now no pus in either ear, but there is some secretion in right ear from the granulating surface which has not yet epidermized over. I will lay in several Thiersch grafts tomorrow, which I think will hasten the epidermization, and I expect this ear to be dry in two or three weeks. The hearing in right ear, he says, is better than it has been for several years. It is watch at three feet, whisper at six feet; left ear, watch at six feet, whisper at ten feet.

LIV.

A MODIFIED RADICAL OPERATION FOR CHRONIC SUPPURATIVE OTITIS MEDIA.*

Based on Eight Cases. Lantern Slide Demonstration of Procedure.

By Hugh B. Blackwell, M. D.,

New York City.

In performing this particular operation on the following eight cases my purpose was (1) to stop the middle ear discharge and (2) to conserve or, if possible, to improve the hearing.

All of these operations were performed in the service of Dr. Robert Lewis at the New York Eye and Ear Infirmary, and Dr. A. B. Duel at the Manhattan Eye, Ear and Throat Hospital.

Operation.—The usual postaural incision is made, as in the Stacke operation. The soft parts anterior to this incision are elevated and retracted forward; the cortex is removed with a gouge and the subcortical cells with curettes until the antrum is opened. The posterior bony canal wall is lowered and the antrum widened to its fullest possible extent. When the short process of the incus becomes visible, the external attic wall is removed by placing the back of the curette external to and in front of the incus and curetting from within outward. The bony canal wall is still further lowered until the facial ridge is reached, leaving only an epitympanic ring in its superior portion, with a width of about one-sixteenth of an inch. In four instances I have removed this ring in the superior and outer quadrant of the circle, leaving the membrana tympani and ossicles intact.

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*Read before the New York Academy of Medicine, Section on Otology, April 14, 1916.

The granulations, polypi, and cholesteatoma lying in the external and internal attic are removed by curetting internal or external to the incudal body, care being observed not to destroy the suspensory ligament of the malleus or its external lateral ligament. In curetting near the incus, great care must be taken not to disturb the ligament which binds the extremity of its short process to the bone below and just in front of the external semicircular canal. The drum and ossicle, of course, are not removed but are left in situ.

An L-shaped meatal flap is cut, as in a radical operation, the cartilage is removed, and the flap sutured to the temporal fascia. The mastoid wound and attic region are packed snugly, thereby furnishing support to the flap, and the posterior wound is sutured.

Case 1.—A subacute condition; history of seven weeks' discharge. Operation three years and four months ago; large perisinus abscess with perforation of the premastoid lamina and necrosis of the adjacent posterior bony wall. The ear has been dry ever since shortly after the operation. The incus was removed.

At present, the patient hears an acumeter at six feet and a moderate whisper at six feet.

Case 2.—Ear had been discharging for two years. Operation was performed two years and nine months ago. Cholesteatoma was found in the antrum. Ear has been continuously dry ever since shortly after the operation.

Whisper heard at eighteen inches, and watch only on contact, before the operation. The patient now hears an acumeter at one foot and a whisper at four feet.

Case 3.—Ear had been discharging for two years. Operation was performed two years and three months ago. The ear has been continuously dry since shortly after the operation.

Prior to the operation, the patient heard a whisper at twenty feet. She now hears a whisper at four feet and an acumeter at the same distance.

Case 4.—Double otitis media purulenta chronica. Right ear had been discharging for one and a half years, and the left for six months. Double operation performed two years and four months ago. Both ears are dry and have been so continuously since shortly after the operation.

Hearing before operation: Right ear, acumeter at three inches and whisper at three inches. Hearing now: Acumeter at two feet and whisper at eighteen feet. Left ear: Before operation, acumeter at twelve inches and whisper at one foot. Hearing now: Acumeter at six inches and whisper at twenty feet.

Case 5.—Double otitis media purulenta chronica. The right ear had been discharging for six months, and the left for two years. Double operation was performed simultaneously seventeen months ago. Both ears have been continuously dry ever since shortly afterward.

Hearing at present: Right ear, acumeter at twenty feet and whisper at twenty feet. Left ear, acumeter at fifteen feet and whisper at fifteen feet.

Case 6.—Double otitis media purulenta chronica. Both ears had been running for eighteen years, following scarlet fever and diphtheria. Operation was performed a year ago. The left ear has been continuously dry since shortly after the operation. The right ear has been dry at times, but is discharging at present.

Hearing prior to operation: Acumeter, right ear, three feet; left ear, six feet; whisper in both ears at six feet.

Patient now hears: Acumeter, right ear, eight feet; left ear, two feet; whisper in both ears at eight feet.

Case 7.—There had been a discharging ear for the past seventeen years. Operation was performed nine months ago. The ear has been dry at times, but is at present wet. The discharge is from the tube.

Hearing before operation: Left ear, acumeter at two feet, and whisper at six feet.

The hearing since the operation is the same.

Case 8.—Patient had discharge from the ear for five weeks prior to admission to hospital. Operation was performed six months ago, and showed necrosis of the posterior bony canal wall and perforation of the premastoid lamina. The ear is now dry and has been so for over four months.

Hearing before operation: Acumeter at twenty feet and whisper at twenty feet.

At present it is the same.

SUMMARY.

The eight cases presented represent eleven primary operations, for in three instances both ears were involved and a double operation was performed.

Nine of these operations have resulted in dry ears which have remained continuously so ever since shortly after the operation. In the remaining two cases the ears have been intermittently wet and dry.

The hearing was improved in five cases. In one case there was some decrease, and in two instances it remained the same. When questioned, none of these patients thought they had suffered any impairment in hearing since the operation.

In this connection, it is interesting to note that in Case 8 the hearing was almost normal before the operation. It is the same at present, showing that in this case at least the procedure had no ill effect upon the hearing.

An interesting feature is the development of a secondary tympanic membrane, which in all of these cases has grown across the attic region in the process of healing, completely closing off the attic from the mastoid cavity.

In performing the dressing it is necessary to pack the attic region snugly until it becomes well dermatized; otherwise it will soon become filled with a thick plug of granulations which, if infected, will be apt to discharge copiously, reinfect the middle ear, and prevent its proper aeration. It is probable that the discharge which occurred in two of the wet cases in this series was caused by the failure to properly appreciate the importance of this point.

It will be seen that the best results were obtained in those cases having a history of discharge lasting less than five years. The procedure seems especially applicable in this class of cases, and more particularly so in those chronic cases in which there is good hearing.

I believe that this operation is indicated in cases of aural discharge of several months' standing which have resisted treatment, and where the patient is threatened with the possibility of a long-continued suppuration. I do not think that the presence of a large perforation or the absence of half the tympanic membrane is a contraindication to the operation, as demonstrated by some of these cases.

The important element to be considered in cases of subacute and chronic otitis media, in the absence of constitutional symptoms, is, of course, the hearing. If this be good and the discharge under five months' duration, I believe this operation to be especially indicated.

In cases of exostosis of the canal associated with otitis media purulenta chronica, and in perforation of the premastoid lamina associated with necrosis of the posterior bony canal wall, it should be the operation of choice.

Another point in favor of this procedure is that the danger of causing an injury to the facial nerve is negligible.

In closing, I would say that this operation is not presented as one that would replace the radical procedure: that operation has its own distinct field of usefulness. It is very difficult to compare the results obtained from a radical operation with those obtained from a modified radical operation, save in a very general way, since of course no one could say what the results would have been in any one of these cases had a radical operation been performed.

I am convinced, however, that the functional results obtained in these case compare very favorably with the best which follow the radical operation, and that in the future, as we learn better how to select cases for this modified radical operation, it will yield even better functional results as well as a dry ear.

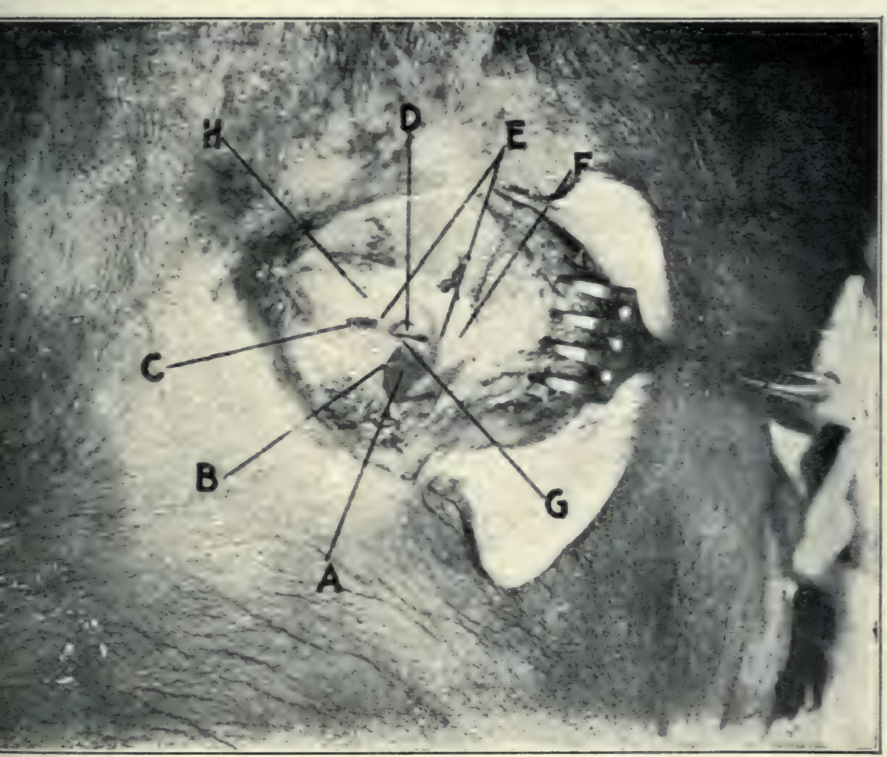

FIGURE I.—View of completed operation, with Epitympanic Ring intact, taken from behind, prior to closing posterior wound. Drum and Ossicles are left intact.
A—Membrana Tympani.
B—Facial Ridge.
C—External Semi-Circular Canal.
D—Malleo-Incudal Body.
E—Attic of the Middle Ear.
F—Plastic Meatal Flap sutured to Fascia of Temporal Muscle.
G—Epitympanic Ring.
H—Antrum.

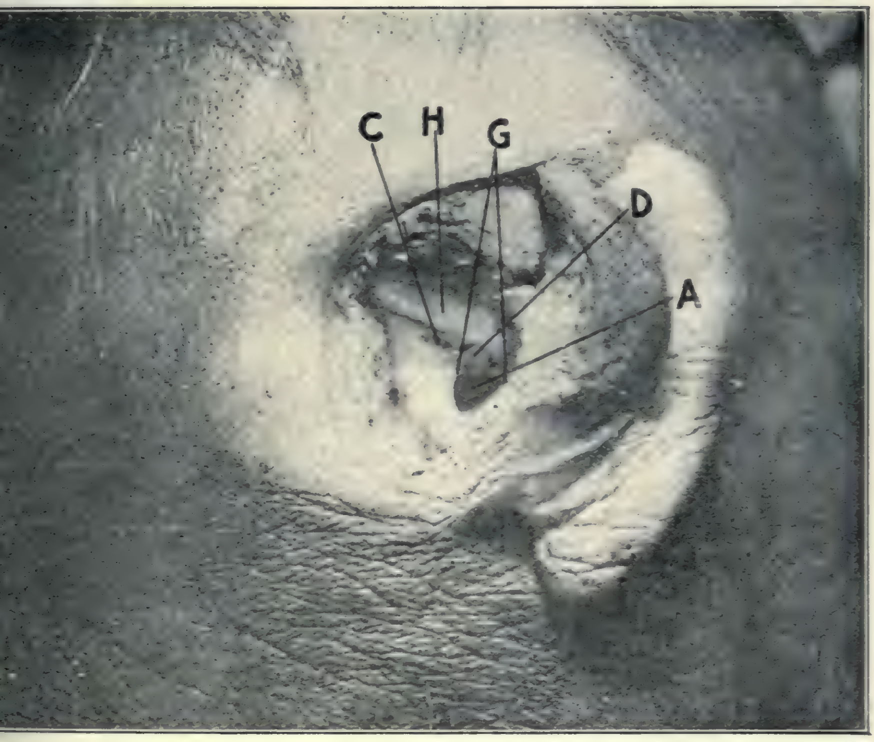

FIGURE II.—View of completed operation, where the Epitympanic Ring of bone has been removed in the superior quadrant. Drum and Ossicles are left intact
A—Membrana Tympani.
C—External Semi-Circular Canal.
D—Malleo-Incudal Body.
G—Epitympanic Ring of Bone has been removed between termination of lines.
H—Antrum.

LV.

NONDIABETIC ACIDOSIS—WITH SPECIAL REFERENCE TO POSTOPERATIVE ACIDOSIS IN CHILDREN.*

By Ira Frank, M. D.,

Chicago.

INTRODUCTION.

It is my purpose in the following paper to bring under one head and in concrete form a review of the literature, clinical and experimental, which is fast tending to outline distinctly the hazy concept we now call acidosis. I shall add thereto certain personal observations and attempt to produce from the whole a practical clinical entity, with definite etiology, definite diagnostic points, specific treatment, and direct and simple prophylaxis.

The subject should be of serious interest to those whose work is related to childhood, and especially to those of us who must frequently anesthetize children—for it is in the young that nonsymptom producing acetone is frequently found—it is in the young that the metabolic balance is most easily disturbed—and it is in the young that we tamper with several of the important etiologic factors of symptom producing acetonemia when we remove tonsils and adenoids under general anesthesia.

The literature on the subject is well nigh boundless, for a study of it leads to many branches and subbranches, each of which terminates in a maze of more or less conflicting theories supported by laboratory and clinical evidence. I consider, therefore, that a brief review of this literature, amalgamating the important points which have been brought out in the course of the last decade or so, and which will assist us in establishing that type of acidosis with which we are chiefly concerned—namely, the postoperative acidosis of tonsillectomies and adenoidectomies—as a definite entity, is in place.

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*Candidate's thesis, read before the American Laryngological, Rhinological and Otological Society, at White Sulphur Springs, West Virginia, May, 1916.

How often the condition occurs in its milder forms few of us probably realize, for the manifestations of the majority of cases are usually overlooked. The well marked cases are less common, but the clinical picture once seen is not likely to be forgotten.

That acidosis does occur more frequently following tonsil and adenoid operations is perhaps due to the fact that children are more frequently anesthetized for this purpose than all other surgical reasons combined. Anesthesia is doubtless the most important causative factor involving, as it does, the starvation of the young patients previous to operation; the destruction of tissue due to the lipoid solvent properties of the usual anesthetics, ether and chloroform, and the modification of the carbonic acid content of blood during administration. In addition to anesthesia, another important element possibly accounts for this relatively frequent occurrence of acidosis in children, namely, that chronically infected tonsils and adenoids *per se* may have a more or less important bearing in the production of acid bodies.

DEFINITION.

Acidosis is a disorder of metabolism—principally of fat splitting, and perhaps to a much less extent protein splitting elements—occurring in the course of, or following, a large number of clinical conditions, characterized by the symptoms of varying degrees of intoxication by acid bodies, and the presence of these bodies or corresponding substances in the urine and blood.

ETIOLOGY.

The occurrence of acetone in the urine of nondiabetics without symptoms referable to this condition is very common, especially in children, and the number of disorders in which it is found is growing steadily in those clinics which require a test for acetone in routine urinalysis. Indeed, in apparently normal individuals under normal conditions twenty-four hour specimens of urine have yielded acetone in amounts up to .02 gram (von Jaksch, Langstein and Mayer).

Bearing in mind then that normal human beings in the course of ordinary economy may show the presence of acetone in the urine, and remembering also that age is indirectly proportional to the ease with which the equilibrium of metabolism may be disturbed, it is logical, on purely clinical grounds, to assume that in a given number of children, certain individuals are more or less disposed to acidosis. In other words, we may consider an "acid diathesis" as an important predisposing factor. In a child with an acid diathesis we would expect to find an acetonuria following minor indiscretions in diet, during the course of mild infections (i. e., coryza); in chronic affections, as chronic tonsillitis, adenoids, chronic appendicitis; and in this type of patient we would expect to see acute acidosis, or the more severe form, acid intoxication, develop from exciting cause which in a normal individual would produce neither symptoms nor signs, at least, not more than acetonuria.

That acidosis is common in children with diseased and hypertrophied tonsils, adenoids, and catarrhal conditions of the upper air passages has long been known. Griffiths, E. Smith and others have noted the acute coryza which is frequently a premonitory symptom of the acute recurrent or cyclic attack. These authors have noted marked improvements, and cures following treatment of the pharynx and nasopharynx. Sedgwick reports a series of twenty-two cases of recurrent vomiting, twenty of which had adenoids or enlarged tonsils, and in most of them there were symptoms of infection preceding or during the attacks. In seven of his cases symptoms of appendicitis were present, two of which were operated with absolute cessation of symptoms. Comby found appendicitis in approximately fifty per cent of his cases.

From my own experience, I can affirm a possible correlation between the chronically inflamed tonsil and acetonuria. We have, as a clinical experiment, operated on a large number of children under conditions almost ideally prophylactic against acidosis, and whose anesthetic has been nitrous oxid for a period not exceeding two minutes. Two-thirds of these cases showed an acetonuria in the first twenty-four hours postoperative, although tests were negative in all the cases before operation. In other words, active prophylaxis, including a remarkably short ether free anesthetic, does not prevent a mild acetonuria in the majority of cases.

Nondiabetic acetonuria and acidosis occurs in chronic inflammations, acute specific infections, gastrointestinal and systemic disturbances. Variation in normal diet, as starvation, deprivation of carbohydrates, maldigestion, or the ingestion of fats in large quantities produce the phenomena. It is often noted in cases of poisoning and in toxemias such as occur in pregnancy and advanced malignant disease. It occurs very often in the course of cardiac and renal affections.

Anesthesia, however, is the most common etiologic factor. Children under twelve years never fail to show acetone in the urine in the twenty-four hours following a general anesthetic. It is to this type I wish to call your attention, as I believe that a study of it will assist in the prevention, recognition and treatment of the symptom producing forms of acetonuria which we term acidosis.

PATHOGENESIS AND PHYSIOLOGY.

The economy of the animal body requires for its life and activity the consumption of fuel. Whether this fuel is supplied by ingested food or by the tissues of the organism, its combustion is essential to the phenomenon of life itself. The source of energy for all physiologic functions is known to be the chemical breaking down of alkalis and bases.

Fats, carbohydrates and proteids produce energy then by a complicated catabolism which results in end products of both acid and basic structure. The alkali radical is life sustaining—the acid principles are life destroying. Metabolism, therefore, necessitates the use of alkalies and the rapid elimination of acid end products—for life is incompatible with acidity.

The acids to be eliminated are in gaseous form or in solution. The gaseous acid (CO_2) is given out by the respiratory act. The acids in solution are thrown off by way of the kidneys and sweat glands—and under normal conditions in such form as to cause no injury to the organism.

When, however, this metabolism is deranged the food elements are improperly or incompletely broken down, and abnormal acid end products result. It is certain of these products, acetone, diacetic acid, and betaoxybutyric acid, with which we are concerned.

In the list of conditions during or following which acidosis occurs, it is evident that in a majority of them a toxic influence precedes the actual formation of acid bodies. For instance, in the specific infections and in inflammations, bacterial toxins and toxic absorption are present before acetonuria is observed. That this is no uncommon forerunner of acidosis is shown by Reiche's report of 3,826 cases of diphtheria and acute anginas, of which 3,200 were acute diphtheria, with varying degrees of acetonuria in sixty per cent. Similarly, a factor of "primary" intoxication is discernible in the acidosis ocurring in Bright's disease, the cachexia of malignancy and the toxemias during pregnancy. In the type of acid intoxication described by Parke and Abt during the weaning period, the primary toxic element may be accounted for either in the artificial feeding or by some quantitative or qualitative change in the mother's milk as the period of lactation nears its close. In the cases of poisoning from phosphorus, salicylates, etc., we have, as toxins, not only these substances per se, but also the toxins produced by the effect of the substances on the tissues.

The use of general anesthetics introduces toxic substances into the blood which vary greatly in their effect on the various organs and tissues. Chloroform may produce serious, extensive, even fatal lesions in the liver, and this, too, following relatively short anesthesias. Ether may affect the liver—but its principal point of attack is the kidney. In anesthesia, therefore, we have also a primary toxemia to account for the frequent occurrence of acidosis. Waldvogel has concluded that a direct toxic action of the anesthetic is responsible for the acidosis in children under his observation, and similar conclusions may be drawn from the recorded series of Rice, Frew, Chalfont, and others.

The mechanism by which acidosis is produced from a primary or preexisting toxic element is shrouded in mystery, but the balance of evidence is in favor of an hepatic disturbance (Embden) resulting in abnormal processes of metabolism, the end products being the acid bodies. McClean expresses this disturbance as the result of the cumulative action of absorbed toxins in the liver, with consequent failure of the organ to properly perform its functions. There follows disarrangement of the carbohydrate metabolism, and the resulting interference with the glycogen storing or glycolytic power of the liver reduces the proper supply of energy producing sugar, thus inducing a disturbance of fat and protein metabolism. As Nasse puts it, the lack of carbohydrate necessary to activate oxygen in the body thereby restricts the oxidation of fats and proteins to their normal end products.

We must assume the acetone compounds to be responsible for the clinical condition we know as acidosis. Betaoxybutyric acid, diacetic acid, and acetone are found so consistently in the blood and urine of these patients, and are so constantly in proportion to the number and degree of the symptoms displayed, that we have for the present no alternative but to believe that the condition is an intoxication or saturation of the tissues and body fluids by these acid bodies.

It has been shown that the three substances, which are structurally closely related, may be produced in the test tube from sugars, proteins, or fats, but clinical observation and metabolic experiments seem to show that the acid bodies are produced from body fat or food fat almost entirely—and possibly, to a very small degree from proteins. The rôle of carbohydrates is still uncertain, but it is understood that sugars are necessary to the economy, either to spare the body fats or to facilitate the destruction of food fats past the acetone stage, because sugar starvation results in acidemia.

The acid bodies in acidemia do not, so far as we can now determine, change to any marked degree the actual alkalinity of the blood, and they are practically nontoxic when administered to healthy subjects in large doses. They do, however, by their slight acid action withdraw alkali from the tissues, and when present for a greater or less time produce nephritis.

PATHOLOGY.

Those cases of nondiabetic acidosis which have come to autopsy show little that can be termed characteristic. Frequently fatty degeneration of the liver is noted, occasionally nephritis. Recent observers (Lucibelli, Crile and others) have found histologic changes in the central nervous system, amounting to a peculiar swelling and liquefaction of nervous tissue.

The constant finding is the marked increase in the blood of acetone compounds, which are present normally in very small amounts. It has been shown, too, that the CO_2 tension of the blood is indirectly proportional to the degree of acidemia.

Though the urine is said to show frequently a rise in ammonia content preceding acidosis, the first practical clinical finding is the presence of acetone as determined by the Lange (Legal or LeNobel) test, or the iodoform test. Acetone is the first of the acid bodies to appear in threatened acidosis, and is followed by diacetic acid, and finally betaoxybutyric acid. As the condition subsides, the substances disappear from the urine in the reverse order. In postoperative acidosis this characteristic aids in the diagnosis of the degree of intoxication and assists in forming a prognosis. With experience it is possible frequently to determine the turning point of an attack in the test tube even with these qualitative tests, before it is clinically evident.

SYMPTOMS.

The first clue to an impending acidosis following an anesthetic is practically always to be found in the pulse record in the first hours succeeding the return of the patient from the operating room. Depending on the duration and depth of the anesthesia, and on the amount of blood lost during the operation, we expect in a greater or less number of hours either a return to normal of the pulse rate, or definite signs that such return is in progress. When in the early postoperative period the pulse rate increases, remains stationary, or lowers only slightly from the rate during anesthesia—and this, too, in the absence of signs of shock, collapse and hemorrhage—an examination of the urine reveals the presence of acetone in marked quantity.

Routine postoperative urinalysis has demonstrated the presence of acetone in a high percentage of all cases, and in children the finding is practically constant. In the majority of cases no symptoms referable to this condition are apparent, and the last trace of acetone disappears under suitable diet in one to three days. In those cases, however, where the pulse rate is high following operation, we have the first intimation confirmed by urinalysis that the patient is reacting to the presence of acid bodies.

I have found that in the great majority of cases this is the only symptom displayed and is followed in one or two days by a return to normal and a gradual diminution in the acetone excreted.

In a second group of cases a persistent high pulse rate is complicated in four to eighteen hours by the appearance of languor and drowsiness, in some instances approaching stupor, and a marked tendency to restless sleep. These patients show an increased respiratory rate, varying indirectly, according to the age of the patient, and directly to the degree of the reaction, from twenty-eight to forty-eight per minute. The respiration is regular, rather superficial, and the odor of acetone—sweet, "fruity"—is noted in the exhalation. The facies is either pale or (if the temperature is high) flushed, with usually a tinge of cyanosis. Dark circles appear under the eyes. The tongue is usually heavily coated .

The stuporous condition may vary from a mild degree to a truly alarming display. It is distinguished from the holdover effects of anesthesia by the fact that the patient has already come out of the anesthetic—in other words, has had an awake period before the onset of this secondary drowsiness. The patient is restless and exhibits more than ordinary postoperative thirst, and if sleep occurs it is only for short periods. Thirst is the principal complaint—even the youngest children are quiet, arousing themselves only to ask for water.

The examination of the urine from these patients reveals a heavy acetone reaction, and a definite trace or positive diacetic acid test. Under treatment, and coincident with the disappearance of the drowsiness or stupor, the diacetic acid disappears, and, more slowly, the acetone reaction lessens in degree, the test becoming negative in four to six days.

In a third group of cases, which is decidedly small (occurring in my practice is about two per cent of operated children), a more severe degree of acidosis occurs. In this type the rapid pulse and stupor is soon followed by frequent retching and vomiting—nothing is retained by the stomach. On entering the room we are immediately impressed with the fact that the patient looks sick. Prostration is marked, stupor is a prominent feature, and the cheeks are flushed, frequently cyanotic. The periorbital tissues become rapidly sunken, and the acetone odor of the breath can be detected some distance from the patient. Nervous phenomena may be present, varying from the usual restlessness to delirium or convulsions—even coma.

Analysis of the urine in this group of cases shows the intense acetone reaction, and the test of diacetic acid is markedly positive. Under treatment we find an early disappearance of the diacetic acid, the test becoming negative in twenty-four to seventy-two hours; coincident with and following this, a gradually diminishing acetone reaction. As these substances leave the urine the patient returns to health by reversing the order of the stages we have enumerated. Vomiting is the first symptom alleviated, followed closely by a disappearance of stupor and drowsiness, and finally there is a return to normal respiration and pulse rate.

Levison has classified the degrees of postoperative acidosis as

1. Subacidosis—acid products without symptoms.
2. Acidosis—definite symptoms, vomiting and marked restlessness.
3. Acid intoxication, with a clinical picture marked vividly by delirium, coma and convulsions, preceded by a stage of restlessness and vomiting.

According to the cases I have seen, it seems more practical to me to consider Levison's first type as a purely physiologic reaction of the tissues to ether, and my three groups of cases, as subacidosis, acidosis, and acid intoxication, respectively.

Chalfont, in a study of seven hundred anesthetics, lays emphasis on the fact that postoperative vomiting is related to acetonuria—i. e., the liability to vomiting is increased when acetone is present. This is no doubt true, but in most cases we are able to distinguish the vomiting of anesthesia from that of acidemia, inasmuch as the latter appears, as a rule, several hours after the anesthetic vomiting has ceased.

TREATMENT.

From the early opinions regarding the nature of acidosis it is readily seen how alkali therapy came into repute, and it is only in recent years that reports regarding the efficacy of alkali therapy, especially in the acidosis of diabetes, were more replete with failures than successes. In the nondiabetic acidosis the successes reported may be due to (1) a moderate alkalinizing effect from such therapy, or (2) the fact that the attack itself was subsiding when the treatment was instituted.

The treatment of postoperative acidosis as now practiced is eminently successful and satisfactory, and consists of (1) the combating of the abnormal physiologic processes which we know are producing the symptoms, and (2) symptomatic control. Prompt reaction to such treatment is the rule, twelve hours being freqeuntly sufficient to change an appalling clinical picture to practically normal health.

To oppose the process we must, according to the laboratory confirmations of our present theory of acid intoxication, supply either the carbohydrate which is lacking in the metabolic mechanism, or some easily absorbable substance which will produce energy sufficient to insure the proper oxidation of the fats or spare the body fats.

Glucose has been most widely used, given by mouth in varying quantities in the mild cases, where the stomach is retentive. In those cases in which the stomach is irritable, rectal feeding of a five to eight per cent glucose solution in normal saline by the Murphy drop method—or, more preferable, interval feedings of four to eight ounces of the solution—results are prompt and gratifying. Subcutaneous injection of sugar solutions have been made with varying results. Levison reports that in his experience subcutaneously introduced sugar is eliminated, without change, in the urine. The intravenous injection of sugar causes a prompt and marked change in the clinical picture, though the procedure is followed by a chill and sudden rise in temperature. None of my cases has been sufficiently severe to warrant this method, but it has been extremely successful in certain cases reported to me by several of my surgical colleagues.

Though glucose alone in the above methods of administration has proven successful from all aspects, it is my practice to supplement the treatment with solutions of sodium bicarbonate, either by mouth or per rectum, with the sugar. This is done not only to gain the benefits it may have in alkalinizing the acid bodies, but also because of a noticeable good effect on the nausea and tendency to vomit.

Glucose is given as the carbohydrate of choice, because it is presumably rapidly absorbed as such. Allen has recently suggested the rectal feeding of dilute solutions of alcohol in diabetic acidosis, which has been tried with success, but I do not believe the prompt action and relatively unirritating character of glucose solutions require us to make any change in our present methods.

As to symptomatic treatment little need be said, for under proper care the more severe symptoms are soon alleviated. When vomiting is very frequent and distressing, simple lavage with plain water, saline, or sodium bicarbonate solution brings relief for four to six hours, and it is rarely necessary to perform this more than twice.

When dehydration has been severe, it may be advisable to give saline infusion. It is certainly of advantage to push fluids at all stages of the attack.

As soon as the stomach permits, a diet consisting largely of soft carbohydrates—cereal gruels, corn starch preparations—is given, and as symptoms and laboratory findings subside, the diet is gradually brought to normal.

PROPHYLAXIS.

Our discussion of both the clinical and experimental sides of acidosis has revealed a number of points which, when brought together, constitute a definite symptom of prophylaxis.

We have seen that certain children are apparently predisposed to acidemia or acidosis—or, as Silberstein expresses it, "an idiosyncrasy with intolerance" probably exists. In these cases it is frequently found that traces of acetone are present constantly, or with slight constitutional disturbances. In general, then, it is possible to minimize the dangers of a tonsil and adenoid operation in these children by routine examination for acetone in the urine, and to reduce the possibility of acidosis in all cases by the reduction of fat, and increase of carbohydrate intake on the day preceding the operation, by the introduction of glucose, either as candy or in lemonade, at this time, and by avoiding the unnecessarily long starvation period in preparation for the anesthetic.

The choice of anesthetic has probably little effect. I have, as previously stated, performed a number of tonsillectomies under nitrous oxid, and have found acetone in the postoperative urine, without, however, any symptoms of acidosis.

Levison states that the drop method of giving ether has reduced the number of cases by about one-half, and in Crile's method of anociassociation with the very minimum use of ether, acidosis is practically negligible.

After the operation the granting of water in rapidly increasing quantities serves to dilute toxic substances, stimulate urination and the elimination of toxic bodies; and the early administration of some form of carbohydrate greatly reduces the number of symptom producing acetonurias.

In concluding, I wish to repeat briefly and emphasize certain points brought out in this discussion which are of practical value to us in that they lead to an early recognition and a promptly successful treatment of a complication of tonsillectomy which would otherwise be a severe strain and cause of anxiety to the surgeon and all others concerned.

First.—Acetone is found in the postoperative urine of practically all children. This acetonuria produces no symptoms in a great majority of cases.

Second.—Varying degrees of acidosis or symptom-producing acetonemia follow anesthesia in about twenty per cent of cases in children, in individuals predisposed by chronic infections, age, etc.

Third.—The symptoms of acidosis are such that three degrees of severity are readily apparent. These are:

(a) Subacidosis. Acetonuria and rapid pulse.

(b) Acidosis. Acetonuria, diacetic acid in the urine in small amounts, rapid pulse, drowsiness, languor, rapid, shallow respiration.

(c) Acid intoxication. Acetone, diacetic acid is markedly present, rapid pulse, drowsiness, nausea and frequent vomiting; heavy acetone breath.

Fourth.—Acidosis may be prevented, or its degree lessened, by a carbohydrate diet preceding the operation; by shortening as much as possible the starvation preparation; by the use of nitrous oxid, or a short ether anesthesia, and by the use of further carbohydrate diet as soon as the patient is in proper condition following operation.

Fifth.—Postoperative acidosis in any stage readily responds to solutions of glucose, administered by mouth or per rectum, or in very severe cases intravenously, with sodium bicarbonate as a possibly valuable adjunct.

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LVI.

THE SOCIAL AND ECONOMIC ASPECTS OF DEAFNESS.

By A. J. Amateau.

New York.

The title of the subject assigned to me for treatment is so sweepingly general, that I should not do justice to the matter at hand without a word of explanation. As I understand, I was requested to deal with the social and economic problems of the deafmute as evidenced in my experience as the manager of the Society for the Welfare of the Jewish Deaf. This paper will, therefore, confine itself strictly to our experience and will deal only with the Jewish deafmutes of New York. Of course, deductions and conclusions can be made as to the economic and social aspects of a similar group of deafmutes, here or in any other city, under the same or similar circumstances.

There are over ten thousand deaf and deafmutes in New York City, twenty-five hundred or more of whom are Jews. This society was organized in 1910-11 to furnish industrial education and secure work for the unemployed Jewish deafmutes of New York; to promote their social and intellectual welfare, and to provide them with religious instruction and opportunities for public worship.

You will notice from the objects that I just read that "industrial education and securing work" were placed first on the list of activities as a means of bettering the condition of the deaf, so that instead of beginning to treat our subject from the social point of view, I shall begin with the economic; for the economic situation of a man, whether handicapped or otherwise, has a great deal to do with his social life. The work was, therefore, started with the establishment of an employment bureau. This employment bureau, by the way, is the only or-

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ganized employment bureau for deaf in the world, and the only place where a systematic study of the deaf from an industrial standpoint can be made, and we have used it as our laboratory.

What is learned there is used in other fields, all in the attempt to make the deaf efficient economic units and to coordinate their life with the larger life of the normal. Through it not only have we placed deafmutes in positions and made them self-supporting, but we have also ascertained to a certain degree what other activities had to be instituted in order to ameliorate the situation of the deafmute. Through it we ascertained their social and intellectual status, and we found that the methods of education employed at the school which they attended, the kind of training they received, their religious training or lack of religious training, as the case may have been, had a great deal to with their social and economic well being. In this way not only did we obtain information as to what was lacking, but we also learned how to proceed and how to measure the effects of those activities that we had already begun.

Through it was found the necessity of creating evening classes for immigrant deaf who, because of their ignorance of even rudimentary English, were unable to either secure jobs or to hold on to those obtained for them. The results have been very satisfactory.

Through the Bureau, it became evident that evening classes should be formed to round out the unfinished education of those of the deaf who had left before the completion of their school course. Over 66 2/3 per cent, or to be exact, one hundred and eighty-five out of two hundred and seventy-five of the cases handled by the Bureau in the first two years were of this class. They are among those who possess an inability to keep their positions. They are what we called our "recurrent cases." They require an expense of energy far out of proportion to their number, and after the energy is expended, there is no assurance that the work done will not have to be done all over again. Part of the reason for their "chronic job losing" is their incomplete education. We do not blame the deaf man, but we do blame his lack of education. Two got eight jobs; one, seven; three, six; eight, four; forty-seven, two, and one hundred and thirty-seven, one. And to avoid this duplication, triplication and even further multiplication of our work, the Bureau has led us to inaugurate plans for evening classes to eradicate some causes for such a chronic job-losing disposition.

It was found through the Bureau that many had lost their jobs because of unhealthy moral and social surroundings. After work they used to congregate about street corners or in dingy rooms back of saloons or in houses where games of chance were indulged in, not to mention other things. One instance of this can be cited:

J. K. was a jewelry polisher making from fifteen to twenty dollars a week. He found that he could earn more by gambling. He quit his job. Later he persuaded another deaf-mute to do the same, but unfortunately C. L. was not a proficient gambler. He applied for work at the Bureau. His case was investigated. His former employer, a crude, uncultured but big-hearted man, was indignant at C. L.'s leaving, protesting that C. L. had no reason to leave because of working conditions in his tailoring and pressing shop. (He had the corner cop, the street cleaner, the gas inspector and the next door barber confirm his statement.) He told us that J. K. had probably something to do with his leaving. This we later found out to be true, and also that some other deaf were being affected.

We felt that the real reason for gambling of some of the deaf, their frequenting of street corners, etc., was not perverted impulses, but rather a misdirected expression of gregarious instincts. To redirect aright these fundamental instincts, social clubs were formed in Harlem, the Bronx, the lower East Side, Brooklyn and Brownsville. These clubs have instilled in their members a social consciousness which has created a respect for themselves as a class, besides the lesser advantages of team work and orderly parliamentary proceedings. The economic efficiency of many has been thus increased. In a few cases we have firmly and politely informed a chronic job-leaving deafmute, whose activities had been detrimental to his economic efficiency, his good name and that of the deaf, that if he did not mend his ways, we would see to it that he is rejected as a candidate for admission to a social club (if he is not yet a member) or that he be expelled if he is already a member. This threat works because the normal members of his family or his neighbors, being unable to communicate and sympathize with him or to appreciate him, the deafmute must seek the social intercourse of his fellow-handicapped, if he is to enjoy life.

COORDINATING SOCIAL AND RELIGIOUS TO ECONOMIC.

The president of the Hebrew Congregation of the Deaf, the religious department of our work, the presidents of the various social clubs, are informed of undesirable situations, and these in combination with pulpit utterances and editorial comment through *The Jewish Deaf* (our monthly publication in their interest) combine to act as an incessant and powerful reminder to the deaf that they are judged as a class; that the individual transgression is laid at the doors of the collective mass; that consequently extraordinary care must be paid by the individual and extra strong pressure exerted by the group, and that if this is not realized in conjunction with the fact that the economic status of the deaf determines their place in society, they will fall back. Satisfactory proofs of the success of these lines of attack are becoming progressively evident. We have of late been astonished to receive calls from employers who have said: "I have a deaf boy here working for me. He saw I needed more help and asked me to phone you up. If you have any more like him, send them along." To any one who knows the jealousy and suspiciousness based on sensitiveness that unfortunately characterize the deaf as a body, this is remarkable.

The employer, speaking generally, does not want to employ deafmutes. He thinks they are no good. You will note the word "thinks." This attitude is fundamentally rooted in among his stock of prejudices, which he struggles bitterly to preserve whole and not to alter; often in the face of uncontrovertible facts. "It may be so, you see, in the case of such and such a line, but it won't in mine."

This prejudice as to the economic efficiency of the deaf can be traced back to the time when there was ample justification for it—when the deaf were illiterate, ignorant of a trade, incapable of doing aught but begging—a load on society, some one that had to be borne with for sweet charity's sake. But things are otherwise now. As a class the deaf have become economically self-supporting. There should be no reason for refusing to give a deaf man a chance at a job, all things else being equal.

This combating of prejudice against the deaf constitutes the main work of the Bureau. The task divides itself here into two main divisions—the worker and the employer. We can and do exercise a great influence over the worker, as partly outlined above, by a coordination of the social, the educational, the religious with the economic. On the employer we attempt different methods which will be treated further on.

There is one particular problem of interest we were confronted with and which we successfully solved. It is the problem of the "riff-raff," those who don't want to work, those who are parasites, lazy, indolent—the hobo class of the deaf. The measure of the harm they have done to the good name of the deaf is incalculable. Many and many an employer whom we had, after strenuous and exhausting efforts, induced to take a deaf man, has informed us that he absolutely would not take another deaf employe. He had had enough. His unfortunate experience with one had made him deaf to our further pleas for fair consideration of the rest.

That such results should have been caused by the actions of some of the irresponsible deaf is deeply regrettable, and their conduct merits utmost and unsparing condemnation. After all, they constitute only two score or so (about two per cent of all the deaf). Yet in 1914, fifty-seven (forty-seven per cent) of the applicants were given one hundred and forty-three jobs (sixty per cent of the jobs), and in 1915, thirty-eight (thirty-six per cent) of the applicants were given seventy-eight jobs (fifty-five per cent). The smaller percentages evidence the beginning of a new policy inaugurated towards the latter half of 1915, part of whose results are indicated in the smaller percentages. It was found that on the average, seventy-five per cent of the employers of such deaf refused to hire any other deaf. This loss is greater than indicated numerically, by the fact that every employer satisfied means not only one more whose prejudices have been overcome and who will employ other deaf, but also one who, if pleased, will communicate his pleasure to fellow employers, who cannot but be impressed by his direct testimony. In view of such a pressing situation, we decided to withdraw all such trouble-makers from the employment market and to place them to work under our direct supervision, a poster-mounting shop being organized for this purpose. Perhaps the results can best be illustrated by citing a few examples.

There is H. L. He was given and lost eight jobs within three months. His record reads: Discharged for laziness; discharged—inefficient; discharged—fighting; discharged—violated smoking regulations. He is a mental defective. His parents are such in name only. His home surroundings are highly demoralizing. For a time he was in a reformatory. We thought him hopeless, but he was given a job in the shop. He is now one of the most efficient workers of the shop. This was done by a policy of strict and rigid kindness. He had an amusing habit of shaking hands with every one he met, stopping all work to do so. Within a month every policeman, ticket chopper and seller on the route of his errands knew him as the "dummy" who always shook hands. He was told to "cut it out and tend to business." At first he demurred, but a frown of reprobation and a warning finger raised as if to a child subdued him. So zealous a worker is he now that it has been found necessary to prevent him from carrying packages too heavy for him. Last week, when his salary was raised from five to seven dollars, he was so overjoyed at the thought that after all he was of some use on earth, that he ran about the shop in hilarious glee, waving his money about. In his case proper treatment had made a workingman of a formerly useless aggregate of flesh and bone.

Another case is M. L., a young lad of good family and breeding, intelligent, but very hard to suit in jobs. Of eight given him, he left six. He was given a job in the shop. Things didn't suit him. He left. Later he was given another chance, though he was told that if he left again he would not receive any help through the Bureau. This kept him there for a few months until his "wanderlust" broke out again. After a few months he asked to be taken back. This was refused. He appealed to his social club and brought us a letter from the president to the effect that he had settled down, and recommending reconsideration. He was then taken back. He is now a better worker than ever, having earned an increase from seven to ten dollars.

H. S. was given eleven jobs. He seemed to be a fine chap, but it was soon found that he needed a bracing up. He was rankly lazy. To settle his case he was discharged and told to get a job for himself. Perhaps then he would learn to hold on to it. He did. Now he is engaged to be married—an evidence of success.

I. M. was a useless mess, without exaggeration. He refused to believe we were serious when we promised not to give him a job for a year if he left. He left and did no work for a year. He found he couldn't get any himself because of his unprepossessing appearance and general ignorance added to his deafness. After he had learned his lesson he was given a job in another factory, with the warning that if he lost it, we would have nothing to do with him henceforth. This seems to work, and he has, mirabile dictu, kept his job three months already.

Our only excuse for the above methods is that they work. Most of those who had positions in the shop are not there now. They thought that because the shop was under the auspices of an organization concerned in their welfare they could do as they please. Their interpretation as to what constituted their best welfare did not coincide with ours. They left, though warned of the penalties of their attitude, which warning they refused to believe was given in seriousness. They were quickly disillusioned and found themselves thrown on their own resources. This necessity, combined with the force of social approval manipulated through the pulpit, the press and the clubs, have made them obtain their own jobs which they are keeping. Many are making out very well. Last year they composed about eighty per cent of our unemployed list. This year they compose zero per cent.

The difficulties in obtaining work have been largely due to conditions outlined already—incomplete education of some of the deaf, chronic job leavers or the "hoboes," the employers' prejudice against employing any handicapped. To these might be added that the employer rarely thinks of calling up an employment bureau for the deaf. Most often when he does, we are unable to satisfy his requirements, because of the limited number of applicants. The demand for labor is in inverse ratio to the number of applications for assistance. The Bureau is constrained to seek positions in those fields the deaf applicants are experienced in or in which they can possibly engage.

In other words, the work of getting jobs for the deaf is purely that of going out and hunting for them, getting past officious stenographers and office boys and obtaining an interview with employers, trying to overcome unexpressed aversion (they rarely express the truth in such cases), showing them that some of the biggest business houses of the city, like the department stores, mail order houses, cloak and suit companies, shops, factories, etc., employ deaf men to everyone's satisfaction, that two hundred different firms have employed deafmutes through our Buerau. "It's all very well," says he, "but how can we talk to them?" Here the value of the method of education, articulation and lip reading come into play. We tell him of the wonderful methods of oral instruction, and arouse his curiosity in this respect.

In some few cases the employer is so astounded by the fact that a deaf man really can work and speak, that he expresses a willingness to see for himself. This is of course curiosity overcoming prejudice. Others advance objections on the ground of efficiency. We argue that the deaf are not distracted by any noises or the usual temptations that distract the normal worker, thus making him more efficient. "There is ample testimony to prove this," we say. Each line of argument must of course follow the peculiarities of the individual employer. Many show a willingness to listen, but that is as far as it goes. Unfortunately, it is not within our power to ascertain to what extent the employers' excuses or reasons for refusal to employ the deaf are true. It is difficult to separate the chaff from the grain in these assigned reasons.

Another difficulty we meet with is the restriction caused by the fact that the schools teach few trades. Printing, carpentry, millinery, hand sewing and sign painting constitute the main divisions of any industrial value. If we ever restricted our endeavors in these fields, it would result in overcrowding. As it is, such is the situation in lines like printing and carpentry. Our reports show over seventy-five different trades the deaf work in, such as:

TRADES AND SALARIES.

Architectural draughtsmen	20
Bird cage makers	6
Bookbinders	9
Bookkeepers' assistants	8
Bottle washers	10
Operators, finishers, pressers on ladies' and men's garments	7-20
Cigar box makers	14
Chandelier workers	12
Rhinestone setters	25
Errand boys	6
Compositors	13
Jewelry polishers	11
Engravers	15
Die makers	15
Hat framers and rounders	7
Shipping clerks	8
Vulcanizers	18
Wire winders	9
Sales agents	7
Upholsterers	25
Slaughterers	22
Feather workers	7
Porters	7
Leather goods workers	8
Sign painters	12
Linotypers	24
Table gluers	8

It will be noticed that these trades require the minimum of intercourse with either fellow workers or foremen. The work is routine, and once set forth no further instructions are needed. Some of the semi-professions, like bookkeeping and architectural draughtsmen, are very rarely acquired by the deaf, and it has in general proven difficult for us, using every source of influence, to obtain work for the applicant. Such lines, though routine, require some intercourse. The deaf, though they can learn these trades and work at them, find it hard to obtain such positions and to keep them. It is inadvisable to train the deaf in any but those trades requiring repetition of movements or where there is no necessity for repeated instructions.

The salaries paid the respective trades are pretty close to the average paid the hearing, though the maximum limit is far from being the same. The department of any trade commanding the high salaries requires a degree of skill which the employer refuses to permit the deaf worker to acquire. The kinds of work the deaf seem to hold on to best, varies directly with the simplicity and extent of the mechanical and routine features of the work.

Work requiring very close contact with other workers is bad for the deaf, because of the constant friction that arises, often making the deaf man blaze forth in anger. The worker likes to tease. The deaf man is sensitive. The union worker thinks the deaf man gets his job because he has agreed to work under pay, and dislikes him for that. The unions demand that the deaf join them, but after this is done they cry out that the deaf are incapable of living up to their standards. This is unjust and untrue. Sufficient data has been accumulated to disprove their contentions. The deaf do not like to work with other deaf. Often their working together handicaps their efficiency, because they cannot resist the temptation to converse. They cannot work and talk; ergo, they talk.

Some modern tendencies seem to be working to reduce the deaf to a lower level, despite the heroic efforts made by the deaf to prevent this. "Safety First" movements, compensation laws, minimum wage acts, tend at first to act against the deaf. As it is, the employer has no strong inclination, if any, to hiring any deaf person. Add to this unwillingness, the fear (unfounded, it is true) that the deaf are more liable to accidents than the normal, which, if true, acts against the employers' financial interests, and you have there a situation which calls for concern. The minimum wage law will have serious effects on the deaf. These laws are of great benefit to the laboring class, and it is hardly fitting that any opposition to these laws should be aroused because of its harm to the deaf.

It is a matter of pride that the deaf do not work against the enactment of these laws. They believe confidently that they will after a time ultimately vindicate their efficiency. This remains to be seen.

The problem here is essentially a school problem—the fitting of the deaf to meet these new and more stringent industrial requirements. This is the great problem now before the deaf. On it depends their whole economic future, and since the economic is the foundation of an individual's life, on it therefore depends the entire happiness of the deaf.

Trade education of the deaf as it is now taught is not altogether satisfactory. When first inaugurated it was sufficient to meet industrial requirements, but these requirements having changed greatly, and the trade education system changed to a small extent, a gap exists which must be filled in.

The teaching of a trade in school has many disadvantages, due to the difference between the selfishness of the business world and the altruistic atmosphere of the school; the change from the one into the other makes the pupil a derelict on the sea of life until he finds himself; the difference in machinery employed in the factory and the school, the difference in time spent in the limited number of trades, and the limited manner in which these few trades are taught because of restricted facilities of space, money and machinery, which to be kept up to date would necessitate complete renovation about every five years—prohibitory, as is evident.

These disadvantages nullify almost completely the value of such an industrial training of the deaf as a means of livelihood.

A complete change in the system is essential—a change which would embody this principle: that the industrial education of the pupil is to be taught right in the shop itself, under real factory management, regulations, atmosphere and conditions, using the same tools as are used by the laborer in making the finished product, while the theoretic side of industrial education is taught in school.

Here it is important that some consideration be given to the method of instruction and communication of the deaf. As it is known, there are two methods—the oral and the sign. Our experience is that while articulation and lip reading are decidedly industrial assets, communication by signs and the manual alphabet is such a social asset to the deaf, that without it they cannot enjoy life or profit by attending lectures, religious services and socials wherever and whenever they must communicate with others at a distance.

The civil service regulations of this city and state forbid employment of deafmutes. This is unfair, for there are many kinds of work they can do. The United States Government allows the deaf to take examinations in certain departments, with a string attached to it, the final appointment being left to the discretion of an appointer who has a choice of one of three.

To summarize, our activities to better the social and economic condition of the deaf are:

Employment Bureau, the social clubs, the weekly religious service, the evening classes for immigrants as a first step toward an extended evening educational system, the publication of the monthly periodical, *The Jewish Deaf,* the rendering of social service as interpreter and intermediary of the deaf in charity organizations, associations, courts, city departments and governmental agencies. Add to these, contemplated extensions which will intimately affect the hygienic life of the deaf, as well as plans tending to create strong sympathetic bonds 'twixt the deaf child and its parents, and you have a measure of how extensively the Society for the Welfare of the Jewish Deaf can mold the life of the deaf.

LVII.

SORE THROAT CLINICALLY CONSIDERED.*

By Samuel Johnston, M. D.,

Baltimore.

"The schemes of any of the arts or sciences may be analyzed in a sort of skeleton, and represented upon tables, with the various dependencies of their several parts." (Watts.)

Sore throat is defined as a popular term embracing almost every inflammation or morbid condition of the throat. (Gould's Illustrated Dictionary of Medicine.)

To study the subject clinically, as is my purpose, it is necessary to scrutinize the nasal passages and accessory sinuses as well as the oral cavity and larynx. We should also scan the physiognomy of the patient, mark well any changes in the voice tone, and note the odor of the breath, before entering into a more detailed examination of the case.

"In the early ages of medical science the doctrine of physiognomy formed an important part of all systematic teaching, and practically it is no small part of the accomplishment of a skillful physician to be able to recognize readily in any sick person the outward signs which may be characteristic of his malady." (Ord.) The staining of the skin and conjunctivæ of icterus; the flush of hectic; the cyanosed countenance of the subject of laryngeal or tracheal stenosis; the facies of the habitual mouth breather; the notched central incisors of hereditary syphilis; "the typical features of myxedema and cretinism," will serve to illustrate what may be gleaned by the close observer.

Changes in the voice are produced by such a variety of causes that it is only possible to name a few examples: paralysis of the soft palate, defects in the conformation of the palatine arch, swollen tonsils, benign and malignant growths in the nasopharynx, palsies of the vocal bands, laryngeal growths, laryngeal inflammations—all produce voice alterations to a greater or less extent.

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*Read before the American Laryngological Association, at the Thirty-eighth Annual Meeting, Washington, D. C., May 9, 1916.

The odor of the breath is a valuable aid to diagnosis and may direct our attention to certain morbid conditions which otherwise are liable to be overlooked. Profound uremic poisoning, now and then, is accompanied by a distinctive odor of the breath; pulmonary gangrene fills the expired air of the patient with an odor fetid beyond compare. Who is not familiar with the scent of the exhalations of the alcoholic, or of that of the victim of quinsy, or of throat syphilis, of ozena, or necrosis of the nasal bones? These are not all the conditions producing foul breath, the list may be indefinitely extended; but decayed teeth, foreign bodies, rhinoliths and disorders of the digestive apparatus may also be mentioned by way of a supplement.

With a strong light now directed into the widely opened mouth of the patient, we may proceed with the investigation of the case before us.

The lips, gums, teeth, tongue, palate, pharynx, nasopharynx, and lastly the lingual tonsil, epiglottis and larynx, in the order named, should be carefully examined.

Search for any reflexes in the nasal passages and in the external auditory canal, for often here may be found the *origo mali.*

Upon the lips, mucous patches, cysts, tubercular ulcers, cancer, malignant growths, herpes, and tertiary syphilitic ulcers may be found. The gums and teeth will be more thoroughly looked after by the dentist; but tubercular ulcers, tumors, etc., are not uncommonly located on the alveolar processes.

The bleeding gums of scurvy, and the blue line upon the gums indicative of lead poisoning, may be the first suggestions regarding these affections. Cancrum oris, herpes and thrush, commonly, and rarely pemphigus, may be looked for on the buccal mucous membrane.

Affections of the mouth form a chapter of their own. Books have been written upon diseases of the tongue, but that abscess, cancer, papilloma, gummatous infiltrations, cysts, syphilitic and tubercular ulcers appear on this organ, and that beneath it ranula, or perhaps an imbedded salivary calculus, may be found, should be borne in mind.

The uvula may be elongated, causing in some instances distressing cough, or this member may be enormously swollen as in acute uvulitis. It is also the site of specific ulcers, or a benign, notably papillomata—or occasionally a malignant—growth may be located here.

In the roof of the mouth tumors are rare. It is, however, a favorite location for gummatous deposits, and often we see these followed by necrosis and perforation.

The tonsils may be atrophied or hypertrophied. These glands may also be acutely inflamed, followed by abscesses in the parenchyma of the gland, or in the peritonsillar tissue. In the lacunæ, calculi are sometimes imbedded, giving rise to the suspicion of a malignant growth. Mycosis tonsillaris, acute and chronic tonsillitis, diphtheria, syphilis in its various manifestations, Vincent's angina, and tumors, may be thought of in studying the tonsils.

The lips, tongue and palate, singly or collectively, may have their motion impaired or abolished, due to central or peripheral disease.

The examination of the nasopharynx should not be omitted, for besides the usual inflammatory affections of this region—namely, adenoid growths, polypi, etc.—an ulcer, usually of an infectious nature, is found here when least suspected. Retropharyngeal abscess and gumma of the pharynx are revealed as rounded swellings projecting from the surface of the normal pharyngeal wall. In recent years the lingual tonsil has been the subject of much study, but as it is liable pathologically to the same changes as the faucial glands, it will suffice to say that its abnormal growth gives rise to cough, a sense of lump in the throat, etc.

The epiglottis is the seat of acute and chronic inflammations; the former occasionally resulting in edema, though we do also find this a part of general dropsy due to cardiac or renal disease.

The microorganisms of syphilis and tuberculosis, unfortunately, too often find the epiglottis a suitable soil for their development and destructive work.

Sore throat is to a greater or less extent the accompaniment of measles, scarlet fever, influenza, typhoid fever, etc. It is a good rule to examine the body carefully as a whole in all cases of sore throat, for often the throat symptoms are only a local manifestation of a general infection.

I am aware that this list of affections causing sore throat is far from complete; it is, however, sufficient if my efforts stimulate a further study of the subject.

The diagnosis is not usually difficult if we examine the cases systematically, but should any doubt arise it is best to ask for counsel.

In regard to treatment there is no hard and fast rule. Each case must be managed in accordance with conditions present, and general medical and surgical, as well as hygienic and dietetic, principles should be the guide.

Recent studies in bacteriology have conclusively proven that many throat affections are due to microorganisms, consequently antiseptic remedies by preference should be used in the majority of cases, though the galvanocautery, nitrate of silver, iodin, chlorid of zinc, and some of the acids may be required to combat ulcerative and rebellious inflammations. A skilled surgeon should decide the advisability of operative interference.

Sanarelli states: "Numerous species of microorganisms are constantly present in the buccal secretions and upon the moist surface of the mucous membrane of the mouth. Some of these are only occasionally encountered and may be regarded as accidental; others appear to have their natural habitat in the buccal cavity. The nonpathogenic organisms possess various properties; some curdle milk, other dissolve albumin; others, again, give rise to fermentation in solutions of glucose and lactose, or invert cane sugar.

"Of the pathogenic series the bacilli of cholera, typhoid, and diphtheria, the coccus of sputum septicemia, and the diplococcus of pneumonia are the most important. The saliva of healthy persons has the power of destroying or retarding the growth of the above mentioned bacilla, but it constitutes an excellent medium for the development of the cocci."

The story told is doubtless an old one ,but I have thought that to formulate certain conditions producing sore throat and sore mouth would be of some little advantage, and assist in their intelligent diagnosis and treatment.

If my experience in the treatment of affections of the throat and nose has proved any one thing, it is that diseases of these regions differ in no way from similar pathologic changes in other parts of the body, and should reecive the same therapeutic and surgical treatment. Conservatism and mild measures, however, should be the rule for guidance.

LVIII.

EXTENSIVE KELOID OF MASTOID WOUND CICATRIX.*

By Frank L. Ryerson, M. D.,

New York.

The patient first presented himself at the clinic of Dr. Kenefick, at the eye and ear infirmary, about four weeks ago. At the time, as a matter of record, Dr. Dickson took a picture of the condition (Figure 1). The case was presented before the section to show the extensive keloid development, which seems to be quite unusual following a mastoid operation. There was no history of any keloid growth or hypertrophy of any scar tissue in the family.

The patient was twenty-seven years of age, born in Austria, and came to this country at the age of eighteen. He had had typhoid fever, but otherwise had always been well.

Two years ago, after acquiring a severe cold, the patient had a pain in his right ear, and for a short time there was a purulent otitis media. Three months later a mastoidectomy was performed at St. Mark's Hospital. After dressing the wound for about four months, the upper part healed, leaving a small wound open at the bottom, which soon healed. The tumor subsequently began to develop along the entire line of the incision. In the beginning there was an entire absence of sensation to touch, both of the ear and over the mastoid area, but during inclement weather there was a prickling and tingling sensation.

There were no scars upon the patient's body, but there were three or four on the fingers.

The tumor was two and one-half inches in length, one-half an inch wide at the base, and three-quarters of an inch wide at the top. The surface was slightly irregular; the consistence, firm. There was a sharp margin to the healthy skin, and no overlap at the base, excepting a small tip at the lower end.

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*Presented before the New York Academy of Medicine, Section on Otology, April 14, 1916.

About nine months ago the patient received a series of X-ray treatments, the duration of each being about two minutes. The last treatment was given six months ago.

It has been customary to divide keloids into two varieties—the false type, which occurs as the result of scratching, injury, burn, or syphilitic scar, and the true keloid, believed to originate in normal uninjured skin; but the case presented is apparently spontaneous, although it may be the result of a slight injury, as scratching, the occurrence of which is entirely forgotten. The tumor develops in those who show congenital or racial predisposition, and is especially prevalent among the negro race.

Workers agree that a keloid consists of horizontal strands of fibrous tissue which originate from the corium, the long axes of which lie parallel to the skin. The vessels are atrophic or completely absent. Hair follicles, glands and muscles are absent or pushed to the side of the growth. Keloids have no capsule, but merge imperceptibly into the surrounding connective tissue. They generally run a very long course; resolution is possible by simple absorption of fibrous tissue, but the tumor will most frequently recur after excision.

Successful treatment for the removal is not as satisfactory as may be desired. Surgical removal, preceded and followed by the X-ray, has given some good results. Electrolysis, cauterization and excision have been of little avail, as the growth has returned with renewed force and increased size. Partial success has been reported with hypodermic injections of fibrolysin (thiosanimin) and glycerin and water. Perhaps as good a therapeutic agent is the application of radium. A report of twelve cases by Dr. Frank Simpson of Chicago shows some good results. The dosage is determined by experience, and the most insistent keloids have been made to disappear by the more or less destructive action of the radium.

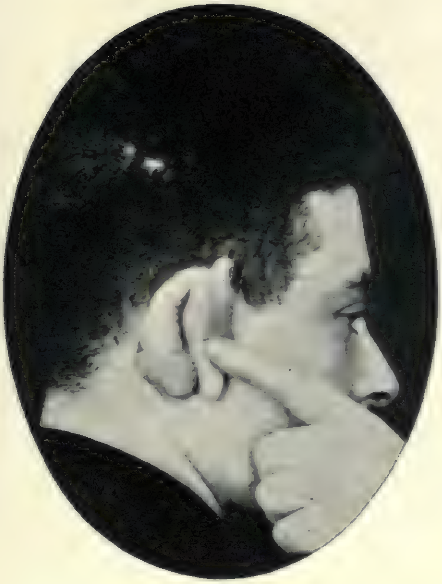

FIGURE 1.

Extensive keloid of mastoid wound cicatrix.

LIX.

CASE OF EPITHELIOMA OF THE POSTERIOR PHARYNGEAL WALL CURED BY THE ELECTROCAUTERY.*

By Dunbar Roy, A. B., M. D.,

Atlanta.

Mrs. R. B. B., aged twenty-seven years, consulted me on July 29, 1913, on account of an ulcer on the posterior wall of the pharynx. Family history was absolutely negative. Personal history: Had always enjoyed good health. No signs of cachexia or physical weakness. Syphilitic history was entirely negative, and this was true of the husband, who was closely questioned. Never suffered with her throat previous to the present attack except an occasional tonsillitis. No history of traumatism of the throat by caustics or otherwise.

For the last three months she had suffered with a throbbing soreness in her throat that was present almost continually. Had been treated during this time by another laryngologist, who used various remedies without success. Finally told her that there was an ulcer which failed to heal and in fact was getting larger all the time.

Examination showed a healthy looking woman with no signs of enlarged lymphatic glands in the neck. On looking into the pharynx there was seen some enlargement of the faucial tonsils, but otherwise healthy in appearance. At the center of the posterior pharyngeal wall, with the upper half hidden by the soft palate, there was seen a perfectly round ulcer almost one-half inch in diameter with a clean-cut border separating it from the surrounding healthy tissue. The whole ulcer was yellowish gray granular looking in appearance and apparently two or three millimeters deep. By palpation the edges were rather firm and elevated, giving the ulcer a somewhat crater shape. It seemed to dip into the pharyngeal aponeurosis. There was some pain on deglutition, but mostly a throbbing sensation. A syphilitic ulcer was the first impression made, with a possibility of its being tubercular in origin. Questioning elicited the fact that she had already been taking mercury and iodids. A piece of the ulcer was removed with Hartman's punch forceps and submitted to a competent pathologist for immediate report. Frozen sections were made and his report was as follows: "The specimen submitted to me shows it to be a typical epithelioma of the mucous membrane."

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*Read before the American Laryngological Association at the Thirty-eighth Annual Meeting, Washington, D. C., May 9, 1916.

Two days later, under cocain anesthesia, the writer undertook to destroy the ulcerative growth by the use of a sharp point of the electrocautery. A deep burning incision was made entirely around the ulceration, about three millimeters to the outside of the rim. All tissue within this ring was then cauterized as deeply and as thoroughly as possible. Sufficient time was taken to see that every point in this area had been touched by the cautery point. There was considerable pain for the first thirty-six hours, after which all discomfort began to subside.

An emulsion of orthoform was given as a local analgesic. There was very little reaction. The cauterized area began to heal and contract so that in two months there was nothing remaining but an area of white scar tissue. This gave no inconvenience and the patient has remained thoroughly comfortable. Only one application of the cautery was made, and at no time did the patient complain of pain during the operation. She was examined about the middle of April, nearly three years after the operation, and there were absolutely no signs of a recurrence.

Unfortunately, clinical observers and writers have been too prone to classify all malignant growths of the pharynx under that general term, without distinguishing between the different forms of carcinomas and sarcomas. For clinical purposes such may be sufficient, but as an aid to their treatment and probably prognosis it is very necessary for the laryngologist to distinguish between these malignant growths whenever this is at all feasible. In the large majority of cases the word "cancer" is supposed to embrace all malignant growths, and unfortunately this term is too often accepted in our clinical reports. This statement is made because in looking up the literature on the subject of this paper the writer found it almost impossible to correllate all the cases recorded, in that many of them were reported in the most unexpected places, and not under any of the headings where one would expect to find them. Furthermore, as Morell McKenzie and others have pointed out, the disease is often so extensive when first examined that it is impossible to tell its point of origin. Since the tendency, whenever found in the pharynx, is to upward extension, malignant growths are apt to be reported under the nasopharynx, tonsils, tongue, esophagus, etc. It is surprising how very superficially this subject is treated in the various textbooks on laryngology. All of the authors seem to be of the opinion that since malignant growths are rarely seen in their early stage, but usually after they have involved the various portions of the pharynx, larynx, and hypopharynx, it is unnecessary to ascertain their true pathology, because so little can be accomplished for the patient. In fact, Dr. D. B. Kyle, who has discussed this subject more thoroughly than any other author of a textbook, has this to say: "The treatment is largely palliative, as no radical operation can be successfully performed." Of course, he means those carcinomatous and sarcomatous growths where the surrounding tissue is infiltrated together with the lymphatics, and where it would be utterly impossible to eradicate the growth. But simple epitheliomatous ulcers may occur in the pharyngeal cavity which can be successfully destroyed by the electrocautery, and in these cases the prognosis is not quite so grave as in other forms of malignant growths.

The writer has been unable to find a case similar to the one here reported. As was stated above, all writers speak of malignant growths, meaning carcinoma as well as sarcoma. The only case found in the literature which is at all parallel is the one reported by Henri Aboulker[1] from the hospital in Algiers. This was a case of epithelioma in a man sixty years of age, which had invaded the left half of the velum, extending to the tonsillar region, to the lateral part of the base of the tongue and to the lateral wall of the pharynx, who was operated upon by pharyngectomy, and who remained cured three and one-half years after the operation. This writer says that in seven out of eleven cases of oropharyngeal epithelioma his results had been successful. He also makes the interesting statement that

Kronlein's statistics give thirty-nine per cent of deaths from operation; Czerny, thirty-nine per cent; Vallas, thirty-seven per cent. Aboulker states that these are practically the only series of such operations which have been published. He believes, in brief, that out of one hundred patients operated upon, sixty survive at the end of six or three months, or even granting that they all die, then they have lived as long as the patients who were not operated upon. Their end is not as horrible as that of patients left to themselves. It is not the intention of the writer to discuss the prognosis and treatment of malignant affections of the pharynx, nor to give any statistical presentation. This has been very thoroughly accomplished in articles by Prof. G. Ferreri of Rome[2] and Chevalier Jackson.[3] The article by Ferreri is very complete and well worth reading, because of his eminently conservative radical treatment of this subject. Under the surgical treatment, this author cites Trelat as declaring that it is useless to intervene in the case of epithelioma of the pharynx. He believes that electrolysis offers the best chances for the patient.

Jackson, in reviewing the subject, has this to say: "Primary carcinoma of the nasal fossæ is rare, yet most rhinologists of considerable experience have seen them. Primary carcinoma of the nasopharynx is so rare, judging from the fewness of reports, that but few rhinologists have ever had one case. A careful search of the various indexes of the medical journals published in French, German, English, and Italian during the last twenty years had failed to unearth more than fourteen cases. It is probable, however, that some reports have not been found; others have doubtless escaped because buried under misleading titles. Cases primary in the tonsil, nasal fossæ, antrum and brain, probably have been reported, if at all, as of those regions. Politzer mentions five cases where the eustachian tube was involved by cancerous extension from the tongue and the superior maxilla, but none of these seem to have been primary in the nasopharynx. It scarcely seems possible that all medical records should contain so few as fourteen cases of carcinoma of the nasopharynx, yet Moritz Schmidt, in a total of 32,997 nose and throat patients, did not meet with one case, though seventy-five of laryngeal carcinoma were seen. Reports of five cases were collected by Bosworth in 1889, to which he added one of his own observation. To these I have added eight, including one of my own, making fourteen in all, tabulating them as well as the incomplete report would permit. The table is incomplete, because five of the cases were evidently not under observation to their termination, and consequently only record symptoms to a certain stage. All of the cases were primary in the nasopharynx, with the possible execption of Latzbeck's, in which the nasopharyngeal portion was discovered at the autopsy."

The treatment is considered under: (1) radical (operative), and (2) palliative (cold snare, cutting forceps, curette, electrolysis, galvanocautery, lactic and nitric acid, "some other caustics," soothing applications, chian turpentine. In only one of the fourteen cases collected was galvanic ignipuncture employed. Death.

Pathologists place carcinomata or socalled cancers under the head of epithelial tumors, because in their structure they are made up of cells of the epithelial type.

They are usually divided into two groups:

1. Acinous cancer with scirrhus or chronic cancer and encephaloid or acute cancer as subdivisions.
2. Epithelial cancer, including squamous or columnar epithelioma.

It is unfortunate that reports of cancer of the throat are not more definite as to the specific nature of the growth. For instance, the encephaloid variety is more malignant and more speedily fatal than the scirrhus. Colloid degeneration of these growths seems to diminish their malignancy. Every now and then an encapsulated tumor is met with, especially in the soft palate (Green), showing no signs of malignancy and yet having the structure of acinous cancer. Then, again, in the variety known as atrophic scirrhus the disease is not uncommonly from ten to twenty years, and the extension only local and glandular.

Epithelioma pathologically is much the least malignant of the cancers, and consequently more amenable to treatment, such as by the use of the electrocautery.

Green in his textbook says epithelioma extends locally, breaks down early and often infects the neighboring lymphatics, but rarely reproduces itself in internal organs. This is probably owing to the size and character of its epithelial elements, which render them much less liable to transmission by the blood and lymph streams than the cells of the other varieties of cancer.

Its malignancy varies curiously with its seat: thus on the skin of the face it has generally a very chronic course and rarely affects even the glands; on the lip early excision gives a fair chance of cure; on the tongue its course is often so rapid, affection of the glands so early, and cachexia and death so speedy, that it must be ranked as one of the most malignant tumors.

In the case here reported the large ulcer on the posterior wall of the pharynx had some of the characteristics of the so-called rodent ulcer sometimes seen on the nose and cheek, and which pathologists place under the head of epithelioma. This case was the type known as "columnar celled epithelioma" or "adenoid cancer," showing also some squamous cells just as transformation of these growths takes place inside of the uterus. These terms are applied to forms of epithelial cancer which grow from mucous membranes of the columnar or cylindrical epithelium variety. They have a great tendency to undergo colloid degeneration. For this reason writers in reporting cases of malignant growths in the throat should certainly try to state exactly the pathologic character of the same, in order that any statistical presentation in the future might be more accurate. Furthermore, we would be in a better position to pass judgment on the final result accomplished by the treatment if we knew the character of the malignancy.

It has now been three years since the case here reported was healed, and there has been absolutely no signs of a return. The results obtained in one case, especially of a cancerous type, certainly does not justify any positive deductions, but the writer believes that the thorough and judicious use of the electrocautery offers the best chance for a good result. In cases where the growth is limited to those portions of the pharynx which are easily accessible, and where they are of the flat ulcerative type, as in the case here reported, it certainly seems to me that such a method would be able to eradicate the growth far better than any cutting operation, especially if the glands and lymphatics were not also involved.

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LX.

DIFFICULTIES AND COMPLICATIONS OF SPHENOPALATINE GANGLION INJECTION.*

BY HARRY L. POLLOCK, M. D.,

CHICAGO.

In a paper read before the North Shore Branch of the Chicago Medical Society last year, entitled "The Injection of the Sphenopalatine Ganglion in Some of the Commoner Diseases of the Nose,"[1] I described the regional anatomy of the ganglion, the technic of the injection, and the indications for the procedure.

Regarding the technic, I stated that if the anatomy is known and the landmarks present, the matter of injection is a simple procedure. I further stated that if the technic is carefully followed there could be no serious complications. At that time there was a complication following one case only, and that was an ecchymosis in the cellular tissue below the eye. Since reporting these cases, I have had several other complications, and have noticed in the literature that others have also had difficulties following the injections, consequently this report.

The indications for the injection of the sphenopalatine ganglion are the same as those I described in the previous paper, with the exception of hay fever. This is because in the past year we have had better results with the use of autogenous pollen in hay fever.

It is my belief that sphenopalatine ganglion injections are indicated in the following three conditions:

1. The true sphenopalatine syndrome, as first described by Sluder.[2] This consists of various degrees of pain radiating to any or all parts supplied by branches of the ganglion. The usual location of the pain is over the root of the nose, in and about the eye, over the frontal region, in the pharynx and tonsillar region, in the ear, back of the mastoid, over the occiput,

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*Read before the Chicago Laryngological and Otological Society, April, 1916.

down the neck, shoulder, the arm, and at times into the fingers. In a majority of cases one or more of these points are involved, but I have never seen a case where all points were affected at the same time.

2. Hyperesthetic rhinitis, which is similar to hay fever, except that it comes on at any time of the year and is irregular in its time of appearance, its duration and severity. Often the paroxysms are brought about by contact with a specific kind of perfume, a particular flower, or the odor from various animals, a slight draft of air, or inhalations of dust. These paroxysms begin with attacks of sneezing, varying in number from twenty to forty or fifty, followed immediately by a nasal hydrorrhea—i. e., a profuse watery discharge from the nose and lacrimation. These symptoms may continue for hours and suddenly abate, only to be repeated on succeeding days, or when again brought into contact with the specific irritant. These attacks may last a few days, or may continue for a week or longer. If the nose be examined between the attacks, the appearance is usually normal except for a pallor of the mucous membrane; but if examined during the attack, the turbinates are turgescent and the mucous membrane red and congested.

This condition has since been described by Sluder[3] as the sympathetic syndrome, an affection of the sympathetic portion of the ganglion controlling the vasomotor and secretory function rather than the sensory. I readily agree with Sluder, except in one particular. He states that there is always a slightly sensory involvement, as manifested by a slight pain or neuralgia. In most of my cases there has been absolutely no pain present, the only symptoms being profuse hydrorrhea and sneezing.

3. Postoperative neuralgia—i. e., where the syndrome or a portion of it makes its appearance only after intranasal surgery has been done—usually following ethmoid exenteration or some work on the sphenoid. In these cases, although the majority were suppurations of long duration, there had been no pain until after the surgical interference.

TECHNIC.

First cocainize the posterior ends of the middle turbinate and the wall immediately behind it, with a twenty per cent solution of cocain. This procedure is also used as a diagnostic measure, and we often find that the neuralgia for which the injection is to be done is immediately relieved. In those cases where relief is immediately obtained, the prognosis from the sphenopalatine injection is always good; but in those cases where not much relief is obtained, but where the pain is sometimes made worse, the prognosis is bad.

After anesthesia is obtained, with a long straight needle transfix the posterior end of the middle turbinate and press the needle upwards, backwards and outwards until the bony wall is felt. This is the anterior wall of the pterygomaxillary fossa, in which the ganglion is situated. Continue the pressure so that the needle passes through the bone into the fossa. This bone is at times very thin and at other times quite thick, so that oftentimes a great deal of pressure is required.

By the pressure sense one usually feels the needle pass into the cavity. The ganglion is usually situated 0.66 centimeters back of the wall (Sluder). The syringe is now attached and a few minims of a two per cent solution of phenol in pure alcohol are injected. If the ganglion has been penetrated by the needle, the patient will at once complain of excruciating pain, radiating from the eye to the ear and mastoid over the occiput and down into the shoulder. This severe pain lasts anywhere from three to forty-eight hours, and is not fully controlled by large doses of opiates. The prognosis in these cases is, however, usually very good, as we know that the phenol alcohol has been injected into the ganglion itself and not into the loose cellular tissue surrounding it.

While the technic as above described is usually quite easily followed, there are certain conditions which make the procedure very difficult.

First—A marked deflection of the septum, a large ridge low down or a split anterior cartilage, would render it very difficult for the needle to transfix the posterior end of the middle turbinate and then be pushed in the right direction. Ordinarily the proximal end of the needle pushes the tip of the nose to the opposite side, but if there is any obstruction, this is not easily accomplished.

Second—In practically all of the postoperative neuralgic and typical syndrome cases where operative measures have already been performed, our most valuable landmark has been obliterated—namely, the posterior end of the middle turbinated body. Many of these cases come to us with the interior of the nose cleaned out and the area occupied by cicatricial tissue, so that the landmarks are not available. Knowing that the point of attack is external to the sphenoid sinus, one can usually be reasonably sure of the correct point if this relation to the sphenoid is borne in mind. Then again, the sphenoid sinus is variable in its size and position, and occasionally one penetrates this sinus and believes that the point of the needle is in the pterygomaxillary fossa.

Third—Probably the greatest difficulty lies in the thickness of the bone forming the anterior boundary of the fossa. In the majority of these cases, if great manual pressure is exerted, the needle will pierce this wall; but I have had cases in which this was impossible. Quite frequently I have resorted to the hammer, and have been compelled to employ great force in driving it through the wall. Care must be taken not to break the needle during this manipulation. There is another objection to the use of the hammer, and that is, one cannot feel the needle slip into the fossa. In several cases I was unable to get through this anterior wall, and explained to the patient that it would be necessary to drill through, but was refused this privilege and was compelled to desist all further attempts.

One of the unpleasant occurrences which I met with was having the phenol alcohol solution run down the pharyngeal wall and into the throat, sometimes into the larynx. This, of course, only occurs when the needle has not penetrated the wall.

The complications following this simple procedure are not numerous, but if it is known that they do occur, much anxiety to the patient as well as to the operator may be avoided. Judging from the literature upon this subject, and from our personal experience, I should say that secondary hemorrhage is the most frequent complication.

Holmes[4] and Sluder[5] both report this condition coming on several days after the injection. The first complication which I desire to report is one of severe secondary hemorrhage.

Case 1.—Miss K., aged thirty-eight years. Referred to us complaining of severe headaches and pain in the infraorbital region, of several years' duration. Has had various kinds of internal medication, and also has had external branches of the fifth nerve injected without material results. A left sided sphenopalatine ganglion injection was advised, and was done March 1, 1915. At the time of injection there occurred an unusual amount of bleeding, but this was easily controlled. When the patient left the hospital on March 2nd there was no evidence of any bleeding. On March 5, 1915, at 9:30 p. m., I was called up by the family physician, who stated that just as the patient was leaving her work at 6 p. m. she had a severe and violent hemorrhage from the nose, which necessitated calling a local physician, who packed the nose. She was carried back to her office, where bleeding continued slowly, notwithstanding the packing. When the patient was brought to the North Chicago Hospital in an ambulance about 10 p. m., she appeared exsanguinated, almost pulseless and still bleeding. I removed the packing and found two small pieces of gauze loosely packed in the anterior portion of the nose. After inserting a postnasal tampon and packing the nose solidly with gauze, the bleeding stopped. The gauze and tampon were removed in three days, with no further bleeding. The bleeding four days afterwards was not due to any septic process, but probably to the alcohol, which caused an erosion of the sphenopalatine artery. This patient was injected again three months later without the slightest tendency to hemorrhage.

In my previous paper I reported a case in which swelling and ecchymosis appeared in the cellular tissue beneath the lower lid, completley closing the eye. This disappeared in a few days without any further trouble.

The second complication which we have had was a paresis of the abducens, occurring within forty-eight hours after the injection.

Case 2.—Miss W., referred by a specialist on July 23rd, 1915, who had had the patient under treatment on account of severe pains in the back of eyes, in both temporal regions and back of ears over mastoid region, also down the arms into fingers. Pain in teeth at times, especially in left upper premolar. All pain more severe on the left side. At one time the pain extended into chest.

Examination showed thorough exenteration of the ethmoidal region. Good breathing space. Sphenoids and frontals open, teeth in good condition, tonsils thoroughly enucleated.

Diagnosis, sphenopalatine neuralgia. Advised injections of ganglion.

On July 23rd my associate, Dr. J. C. Beck, injected the left sphenopalatine ganglion with a few minims of a five per cent phenol alcohol solution. There was not much pain from the injection nor much relief from symptoms.

July 29th. There being no cessation of the symptoms, I injected the same side. I had no difficulty in penetrating the wall and entering the fossa. Injected a larger amount than usual, about ten minims. She immediately had a severe pain in her left eye over her left mastoid and in the occiput.

July 30th. Pain still severe in and about the left eye. The other pain less.

July 31st. Still has pain in eye, and there was noticed a swelling in the infraocular tissue. Complains of diplopia and some blurring of vision. Examination shows paresis of the external rectus, but there is no change in the fundus.

August 2. Vision still blurred. Has had considerable pain in left eye for two days.

August 4th. Pain in left eye. Vision 20/20 in both eyes with her correction, but diplopia is still present; which, however, is corrected with a prism of 9° base out, 6° base down. More pain in attempting to look up. Returned to her home.

August 16th. Received a letter from her physician stating that the pains were the same.

Diplopia continued, requiring a prism of 16° to give single vision.

September 5th. There is an improvement in pains and the external rectus only requiring a 7° prism to overcome.

October 3rd. The eye has entirely cleared up, but general condition just the same as before the injections.

We were at a loss at first to understand the cause of this unusual complication. It was not until just recently that I read Dr. Sluder's latest paper, in which he reported a similar case in which the paresis cleared up in eight weeks and the diplopia in twelve weeks.

With a knowledge of the relation of the pterygomaxillary fossa to the orbital cavity, and knowing that the abducens is the smallest and most vulnerable of all the cranial nerves, I think we can readily explain this complication which we had.

The orbit is connected with the sphenomaxillary fossa by the large orbital or sphenomaxillary fissure. The fossa containing the ganglion is filled with loose cellular tissue which extends into the orbit. As stated before, I employed a larger amount of alcohol than usual, and most probably some of it found its way into the orbital cavity and attacked the small nerves, causing this temporary paresis.

The patient returned December 20, 1915, not having any cessation of the symptoms, for further treatment. Ganglion was injected twice with the same solution as formerly, without any appreciable relief. There is, however, no recurrence of the paresis.

Case 3.—Mrs. C. C. H., aged fifty-two years. June 10, 1915. Referred by Dr. P. For eleven years complained of pain, extending over right side of head and through right eye. This condition gradually became worse. The pain, starting over right eye, radiated over top of head to the mastoid and down into the right shoulder, sometimes going into the throat. Cold or heat does not relieve it. Had nose operated upon six years ago. Sixteen years ago had laparotomy, ovary and tube having been removed. Five years ago had operation on muscle of right eye, since which time she has had diplopia. Supraorbital nerve injected with osmic acid, after which the muscle over eye became paretic. Has had alcoholic injections into infra- and supraorbital nerves, without any relief. Two Wassermanns were negative. Also treated with iodid of potassium, without relief. X-ray pictures show nothing abnormal. Menopause three years ago. Nose and sinuses examined four years ago by specialist, who found nothing abnormal.

Examination of right nostril, normal.

Left nostril showed no evidence of pus or polypi. Slight irregularity of the septum. Throat and ears negative. Five grain doses of aspirin control pain for about five hours, and then it reappears, necessitating the administration of more aspirin.

Diagnosis, sphenopalatine neuralgia. Injection advised.

June 13th. Right sphenopalatine ganglion injected by Dr. J. C. Beck. The pain became more severe, and about five hours afterwards it was necessary to give aspirin for the control of same.

July 14th. Condition the same.

July 16th. I injected the right sphenopalatine ganglion again with one per cent solution of phenol alcohol. Apparently the ganglion itself was injected, as the typical results were obtained. For several days following the patient was very much better, although still not free from all the pains. There seemed to be some slight pains around and in the right eye.

Patient remained in hospital for ten days and then was discharged and told to report in one week.

July 20th. Was called to see patient at her home, in consultation with her family physician, from whom I elicited the following:

Four days after leaving the hospital she suddenly developed a severe pain in right leg, which caused her to go to bed and call the physician. She was unable to bend the leg or even to move it. The diagnosis was sciatica, for which he was compelled to give aspirin and opiates, which only partially controlled the pains in her leg. The pains in her head were all gone, due to the opiates and aspirin.

July 22, 1915. Her condition was improved, although she could not walk without crutches. Was in bed for several months following the time I saw her at her home. The former pains in the head and face are worse than ever.

There is no anatomic physiologic connection between the sphenopalatine ganglion and the sciatic nerve, yet I am forced to include this unfortunate condition as a complication, because of the fact that it came on a few days following the injections of the ganglion, while the primary condition has existed for over eleven years. That the causative agent for the two conditions might be the same is possible, yet from the above facts it must be considered as a complication.

CONCLUSIONS.

The injection of the sphenopalatine ganglion is usually carried out without any difficulties. The difficulties are in a majority of the cases overcome if one is familiar with the regional anatomy.

The complications are not numerous nor severe, but one must be watchful and bear in mind that they do occur and be prepared to meet the emergencies as they arise.

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LXI.

INTRAMURAL MALIGNANT TUMOR OF THE LATERAL WALL OF THE NASOPHARYNX.*

BY EDWARD P. NORCROSS, M. D.,

CHICAGO.

Cases of primary malignant growth of the nasopharynx are of rare occurrence. In the textbooks there is little or nothing mentioned about such tumors. There are numerous reports of single cases, but the description is so indefinite in many of them that it is not possible to ascertain whether the tumor really originated in the nasopharynx or elsewhere. In other reports the diagnosis cannot be considered as definite, because the description of the character of the growth is not exact and permits of various explanations.

By primary carcinoma of the nasopharynx I refer only to those growths that clinically as well as histologically are malignant from the beginning and originated in the nasopharynx. I have, therefore, ruled out of this discussion all malignant tumors of the nose and of its accessory cavities, tumors of the pharynx, primary tumors of the cervical glands, and all tumors of the nasopharynx that are the result of metastases, or the encroachment of malignant growths from neighboring structures.

In reviewing the literature up to the spring of 1913, I found very few reports of primary malignant tumors of the nasopharynx, and especially of primary carcinoma. As late as 1901, Chevalier Jackson[1] reported a case of primary carcinoma of the nasopharynx, with the statement that in the German, English, French and Italian literature of the last twenty years, he could find only seven reports of primary carcinoma of this region, in addition to six cases reported by Bosworth.[2] To these Jackson added one, and of these fourteen cases no two were reported by any one observer, the earliest being that of Durand Fardel, in 1837. Laval,[3] in 1905, reported seven cases of carcinoma observed by himself, and seventeen cases collected from the literature up to that date, the earliest being that of Michaud, in 1845, and nine of which were not cited by Jackson, whilst we find seven cases in Jackson's statistics that are not mentioned by Laval.

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Since the publication of these two papers, primary malignant tumors of the nasopharynx have either increased in frequency or are being more accurately observed. In 1911, Citelli,[4] of Catania, reported ten cases observed by himself, four of which were sarcoma and five carcinoma. In 1913, E. Oppikofer,[5] of Basel, reported a series of twenty-one cases of malignant tumors of the nasopharynx, which he had personally observed since 1893. Strange to say, he observed only three of these during the time from 1895 to 1901, and the remaining eighteen cases since that date. Six of these cases were carcinoma. Oppikofer ascribes this to the progress in the technic of examination.

Trotter[6] reports a series of twelve cases. Compaired,[7] Aboulker, and others, report several cases of their own.

Furthermore, I might call attention to the statistics of Moritz Schmidt, of Prague, that of about thirty-eight thousand nose cases treated in the clinic, not a single case of primary malignant tumor of the nasopharynx was observed.

Since I made the above review of the literature there have been two papers on the subject in the American literature, one by Kelsey and Brown,[8] appearing in 1913, and the very recent report by Dr. W. E. Gatewood,[9] of Chicago, of two cases that were observed in the Presbyterian Hospital in the years 1909 and 1910.

Having called your attention to the infrequency of these primary tumors, and, because they present such definite symptoms, based upon an anatomic foundation, the report of even a single case seems justifiable.

The case I wish to report was first seen by me November 16, 1912. The patient was a young woman, twenty years of age. Her family history was negative, as was also her past history, as to illnesses. She consulted me because of a small gland that had recently appeared at the angle of the left jaw. Her tonsils had been removed two months before seeing me. She did not complain of anything except the gland in the neck. General nutrition was excellent. My opinion was that the enlarged gland was due to absorption from a fragment of the left tonsil, enclosed in adhesions to the pillars. She returned to the physician who had removed her tonsils, and had this fragment excised. I did not at this time examine the nasopharynx.

I did not see her again for a year, during which time many of the deep cervical glands on the left side had become enlarged, and a few on the right side were palpable. She had lost about ten pounds in weight, and stated that she was not hearing well. A diagnosis of probable tuberculosis of the cervical glands had been made, and these were removed on both sides of the neck. The pathologic examination of the glands showed them to be carcinomatous. I had an opportunity to examine her soon after the operation. Anterior rhinoscopy did not show anything unusual. Posterior rhinoscopy showed that the postnasal space was much smaller than one would expect to find it. There was no tumor mass visible, but the left lateral wall looked infiltrated and was hard to the touch. This infiltration extended down the lateral pharyngeal wall. The soft palate was asymmetrical, but did not bulge. There was a moderate degree of middle ear deafness in both ears. She complained of some neuralgia in the side of the face.

Up to this time the patient had not felt very ill. She had not considered her condition serious, and had married shortly after I first saw her. During the year following the neck operation she grew rapidly worse. Nasal respiration became difficult. She lost weight rapidly, and there was marked bulging of the soft palate, with ulceration, and a profuse secretion from the throat, with very foul odor.

She was noticeably deaf—whisper voice was heard only about two inches from the ear. Rinne was strongly negative. The upper limit remained high. The membranæ tympani were congested, and there was absence of light reflex. There was marked anchylosis of the temporomaxillary joint, so that the teeth could only be separated about half an inch. The pain in the forehead, face and neck was so severe as to require very large doses of morphin.

The patient died two years and three months after I first saw her. An autopsy had been promised, but at the last was refused.

Very little can be said as to the etiology of postnasal carcinoma. Most of the cases show that the patient has been of sound and healthy origin, and had not had any serious diseases. It is an interesting fact that carcinoma of the nasopharynx occurs in early childhood. The youngest case that I have been able to find in the literature is the one reported by Oppikofer, in a girl, thirteen years of age.

As to the original site of the tumor, it is usually in the lateral wall of the nasopharynx, although it may arise from the vault and spread to the side. Frequently the case is seen so late that it is impossible to determine the exact origin of the mass. Early in the course of the disease it may only be indicated by inflammation of the lateral wall. At such a time anterior rhinoscopy reveals nothing abnormal, and even posterior rhinoscopy may be apparently negative.

The trouble with hearing is one of the earliest symptoms, and this would point to the lateral wall of the nasopharynx as the most frequent starting point of the disease.

The symptoms of carcinoma of the nasopharynx are interesting, first, because in the early part of the disease there is very often entire absence of symptoms that ordinarily could be interpreted as due to disease of this locality, and, second, because such tumors frequently present a clinical picture of remarkable similarity, due to the fact that their origin in the lateral wall of the nasopharynx involves the inferior division of the fifth nerve, the orifice of the eustachian tube and the levator palati muscle.

Frequently the first symptom complained of is the pain in the area of distribution of the third division of the fifth nerve. Later the pain may spread to the second division. This pain may have existed many months, and the treatment may have been directed to that of trigeminal neuralgia. The pain varies in intensity, but is frequently excruciating and worse at night.

Cases have been reported where the growth has spread through the foramen ovale and involved the Gasserian ganglion, so that we must think of the possibility of a postnasal growth in considering a Gasserian ganglion operation in the treatment of intractable trifacial neuralgia.

The paper by Plummer and New,[10] 1914, concerning a tumor of the middle cranial fossa involving the Gasserian ganglion, is a good illustration of this. It is also possible that the ganglion, which lies near the junction of the roof of the pharynx and the posterior wall, is irritated at the time when the growth is extracranial.

As the tumor originates in the lateral wall of the postnasal space, the eustachian tube is early involved, and middle ear deafness may be the first symptom complained of. Cases have been reported where a transudate formed in the tympanum, or where a suppurative otitis media, with rupture of the membrana tympani, occurred. Oppikofer reports one case in which a mastoid operation was performed on account of the discharge and the otalgia.

Enlargement of the glands of the neck often occurs early, and may be the symptom which brings the patient to the physician. The enlargement of the glands may be out of all proportion to the size and intensity of the primary growth. This adenitis usually appears below the upper insertion of the sternomastoid, and behind the angle of the jaw.

Citelli reports a case similar to the one which I have cited above, in which the enlarged cervical glands were removed before the primary tumor in the retronasal space was diagnosed.

Trotter lays stress upon the finding of some abnormality of the soft palate of the same side. He states that the defect is an inability to lower the soft palate on account of infiltration preventing relaxation of the muscles, and is not due to involvement of the nerve supply. The palate looks asymmetrical, but the patient cannot raise the palate well.

If the tumor proliferates not only into the nasal cavity, but also into the maxillary sinus, there may be severe pain in the area of the second branch of the fifth cranial nerve, and bulging of the anterior wall of the maxillary sinus and the hard palate.

Only a few autopsies of malignant retrotarsal tumors are reported in the literature. Whilst the cervical glands are affected almost without exception in the earlier or later stage of the disease, metastases in other organs are very rare.

The prognosis is absolutely a bad one. Trotter states that he has not observed a single case that lived over fifteen months after the first examination, and this statement is borne out by reports from other clinicians.

In conclusion, I wish to again call attention, first, to the fact that so many of these cases do not complain of symptoms that would be attributed to the postnasal space. In my case nasal obstruction was one of the last things complained of. Second, of the importance in cases of trigeminal neuralgia of making a careful postnasal examination and testing the hearing. Third, enlarged cervical glands may be due to an unobserved carcinoma in the lateral wall of the postnasal space.

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LXII.

FURTHER OBSERVATIONS ON THE RELATION OF AUTOINTOXICATIONS TO ACUTE MEMBRANE DISTURBANCES.*

By Sargent F. Snow, M. D.,

Syracuse, New York.

In 1908 I began a series of articles on the relation of autotoxic states of the system to inflammatory processes. Of this series, in which this is the seventh, two were read in 1909, one before this society, and one before the Sixteenth International Otological Congress at Budapest. In 1912 another came before this society, and during the years 1910, 1911, and 1915, papers before other medical societies, state or national, were read by the author, all calling attention to the intimate relation that appeared to exist between autointoxications and inflammatory activities, so that we may call this paper a "Further Observation" on the Relation of Autointoxications to Acute Membrane Disturbances.

In presenting such a paper, I will try to show as clearly and concisely as possible the conclusions drawn from a twenty-five years' practice devoted exclusively to the ear, nose and throat, twelve years of which has included active dispensary and hospital work, the whole twenty-five years having practically been employed in meeting and combating membrane disturbances. The views set forth need not be accepted as authoritative, for they are largely personal views and may be at variance with some generally adopted, but they are presented with honesty of purpose and the hope that they may help clarify the situation as to the fundamental principles involved in handling at least common head colds and allied inflammations of the accessory sinuses, ear, mastoid, pharynx and larynx.

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*Read before the American Otological Society, Washington, D. C., May 10, 1916.

Let us start with the premise that the human body is a fortress, equipped with munitions of war, and a fighting army; that for the ordinary diseases it has a trained army of body cells, capable of withstanding the onslaughts of an outside enemy, if our body functions are working so well that we have not an inside enemy undermining our resistance.

For the extraordinary diseases, such as smallpox, typhoid, typhus and other acute scourges of mankind, our system seems to require inoculative protection to especially train body cells against those particular kinds of infection, as was so ably pointed out by Professor Vaughn[1], at the last meeting of the New York State Society in Buffalo.

All our respiratory membranes and the membranes lining connecting cavities are the common habitat of various inflammatory bacteria of outside origin, ready for propagation and development as soon as our army of defense becomes weakened or is called upon to subdue some inside enemy. This inside enemy seems to be always with us in the form of intestinal bacteria, believed by Combes, an excellent authority on autointoxication, to be "provided by nature to complete digestive processes." But these same digestive bacteria, under putrefactive influences, may become overabundant and their toxins a menace, unless kept under subjection by inhibiting liver and other bacteriolytic secretions from above.

I, myself, freely accept the belief that we should maintain a proper balance between the inhibitory intestinal juices of the upper bowel and the bacteria of the lower bowel; while such a balance is kept, we need have no fear of colds and other common membrane congestions. In other words, the determining "cause of membrane inflammations" seems to rest upon the question whether our protective army is in full array, or is engaged in a battle with a putrefactive disturbance in the intestinal canal. If it is engaged in such a battle, it leaves our nose, ear and pharynx membranes unprotected and a prey to pernicious microorganic tenants. Concisely stated, intestinal autointoxication seems to permit the development of inflammations elsewhere. An autotoxic state favors acute membrane disturbances—our patients are then vulnerable to outside infective influences.

By autointoxication is meant those self-poisoned states that come from bacteria originating within our body, not autoinfections that come from bacteria originating outside our body.

Some autointoxications are active, resulting in socalled bilious attacks, etc.; some are passive and more chronic, resulting in sensitive cachectic individuals prone to various disorders.

Some autotoxic attacks are only attended by a slight malaise; other attacks are more pronounced, showing fever and a general systemic disturbance. The first mentioned kind is often periodic and is the actual basic cause of many of the chronic diseases of the body, while the more active and profound autotoxic states seem to furnish the groundwork of destructive inflammations.

Sir Arbuthnot Lane, the famous English surgeon, is reported to claim that "most of the ills and diseases that the flesh is heir to are of autotoxic origin, and that were it not for intestinal stasis and autointoxication there would be no need for physicians and surgeons."

In my 1910 article I find the following: "If we are able to handle and prevent acute inflammation of the respiratory membranes, known as common colds, by recognizing and treating the patient's autointoxication, what may we not hope will some time be done with the more grave affections. It appears to the author like the dawn of a new era, and he hardly dares let his fancy travel."

Today, in the year 1916, it would seem that scientific workers, supported by laboratory findings, were bringing my dreams true, and that my earlier articles did rightly predict the dawn of a "new era" in medicine.

The most prominent causes of an autotoxic state are lowered vital powers, overeating, constipation, or at least inadequate bowel movements—bowels perhaps correct, so far as regularity and number of passages are concerned, but the stools are compact or hardened masses that represent what a distended, overloaded colon can no longer retain—a marked intestinal stasis—being a habit rather than the needed free unloading of soft feces two or three times a day. In autotoxic subjects the transition from a healthy state to one of acute inflammation is often but a short step. The toxins that arise from putrefactive changes in a constipated subject permeates the whole system and saps his vital resistance.

It is not irrational to expect that incompetency of the ileocecal valve with regurgitations is a common cause of ileal stasis, as suggested by J. H. Kellogg.[2]

Autointoxication is practically the determining cause or systemic condition that permits inflammations. I say permits, because nature seems to have given us immunity from most outside germs if our functions are working properly.

Quoting from my last paper before this society, published in October, 1911,[3] I note this was said: "Autointoxication has a multiplicity of causes, each one leading up to a time when the system can no longer tolerate its load of poisonous substances. The different organs and membranes of the body are forced to do, and will do, double eliminative work until some organ fails or flags in its function; then we have an acute inflammation in some part of the body from the faulty metabolism—an autotoxic crisis."

Quarantine and sanitation are solving many problems that have to do with our outside enemies, but the problems surrounding questions of diet, digestive function, eliminative organs, and how to keep our inside enemies under subjection are still open to much study and investigation—in fact, they are the pregnant medical questions of the day, and I hope will soon be taken up with more energy than is now displayed.

We are not sure yet whether food should even be cooked or baked. We certainly were not created to gorge ourselves with the conglomerate, but attractive combinations often set before us. It is no wonder that our liver and digestive secretions fail in their task to keep the proper balance. Temperance and caution in eating are quite as essential as temperance and caution in drinking. Overindulgence is quite as disastrous in one as in the other, if we take into consideration the many afflictions that can be traced back to self-poisoning.

I would place special emphasis upon the need of local efforts, surgical and otherwise, towards keeping inflammations within bounds. We must not disregard the knife, nor delay its use, to prevent absorption of pus products, but we are all justified in attempting to avoid operations when the case is seen reasonably early. It can hurt none of us to give a little more consideration to the fundamental causes of inflammatory actions and be a little less led by the glamor of brilliant operating.

When the patient is "ripe with pus" because of bacterial virulence or delay in seeking medical advice, we have no alternative but to secure free and adequate drainage at once; but if he is seen early, before pus formation, we can very properly and safely expect good effects from our "emergency agent," calomel, given freely and fearlessly under twentieth century methods.

The scope of this paper does not permit me to go very deeply into the question of treatment. My personal method in all acute membrane inflammations is to start with calomel, two to four grains, in quarter grain doses, according to the case, repeating it next day if marked activity persists. In mastoid troubles I frequently start with an enema; in fact, I cannot speak too earnestly nor urge too strongly a prompt, efficient clearing of the bowel in all acute inflammations, even if only salts or castor oil are chosen.

The prejudice against calomel has deterred and is even now preventing its use in many cases of acute membrane disturbances where its proper employment, as a stimulant to antitoxic secretions, would give much relief and be likely to save the patient from the knife. My records are so replete with instances where startling and impressive results have followed the adequate use of this form of mercury even in streptococcic infections, before or after operating, that I would urge all to throw aside their fear or prejudice and give it a trial in these dangerous cases, both as an emergency cathartic and antitoxic stimulant.

As early as the year 1909, in the paper of this series presented at International Otological Congress held in Budapest, the following observations were made: "Calomel, we know, not only eliminates autoinfective material, but it disinfects the lower bowel. It also stimulates functional activity of the liver and of the glandular structures of the body, so that their secretions act as effective antitoxins against bacterial invasion and consequent active membrane congestions." The idea that mercury stimulated the production of body antitoxins was the only way I could then explain clinical results, and this has actually been my belief since 1905. Now, seven years later, my attention has been called to the fact that Professor Sajous[4] makes the same claim as to the ability of mercury to stimulate body antitoxins, calling them autoantitoxins, explaining how they are produced, as follows: "The various salts of mercury owe their therapeutic value to the energy with which they stimulate the test organ," which he concludes is the anterior pituitary body. "Only small doses (one-tenth grain) of calomel are needed to excite the test organ, and make the blood autoantitoxic, but if flushing of the intestine is desired and a copious flow of antitoxic intestinal juice in addition to the hepatic action, larger doses (one-sixth to one-half grain) every fifteen or thirty minutes until one or two grains are taken, may be ordered."

He further says: "The bacteriolytic and antitoxic activity which the blood and the intestinal juices acquire under the influence of mercurials accounts for the beneficial effects observed in various infectious diseases."

In cases of active infection, minute doses, given at two to four hour intervals, seem a rational treatment, though, as stated earlier in this paper, I start them with quarter-grain doses, at short intervals, until two to four grains have been given, following this with a saline so as to be ready to repeat the strenuous treatment next day if necessary.

Personally, I do not like to advise calomel or any cathartic, except in emergencies, but I consider the danger from its intelligent use slight as compared to the benefits derived.

With reliable and useful agents to stimulate the production of "autoantitoxins" for the prevention and cure of disease, how much greater will be our leverage, how much better armed we will be, how much the practice of medicine will be simplified, if men engaged in general and surgical work find also, as I have from time to time tried to show, that an autotoxic state is a determining factor in most of the inflammations of the body, both acute and chronic.

All the cases of laryngeal tuberculosis coming under my observation have had what I now know were marked autotoxic disturbances with each exacerbation, and these exacerbations have seemed to be the result more than the cause of their autointoxications. Sajous even contends that the development of cancer is due to hypoactivity of the body's protective mechanism.

To sum up, gentlemen—I will first ask your indulgence—as this paper is probably far from being the "last word" on the subject, but in all candor I must say that since my practice has been conducted along the lines indicated, noting carefully by the skin, eyes, tongue and breath each evidence of self-poisoning, vigorously combating this autotoxic factor and waking up the patient's vital resistance, I find my work much more simple, clear cut and satisfactory. Inflammations commonly melt away like snow under a summer sum, and the need of the knife becomes less and less frequent.

Further observations, making fifteen years that I have worked along these lines, gives me courage to state that I honestly and earnestly believe that when conditions are better studied, the medical profession will accept the fact that autointoxication is responsible for most of our human suffering, and that we have agents so efficient in controlling inflammations, by stimulating antitoxic secretions, that seventy-five per cent of the emergency operations we are now doing will be found avoidable, old age will be defered, and the practice of medicine will have to be largely rewritten.

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LXIII.

A SEQUESTRUM OF THE LABYRINTH.*

By Lester Mead Hubby, M. D.,

New York.

J. B., colored; male; aged fifty years. Had measles when eight years old without ear involvement. Married twice. No children, no miscarriages. Denies syphilis.

Fourteen years ago received scalp wound near vertex over the right ear from a blast in the subway. Noticed deafness of the right ear from that time.

Had chronic purulent otitis of the right ear for five months previous to his attack of mastoiditis.

Early in August, 1915, had furuncle of the left ala nasi, which healed quickly after being opened.

About August 9th had severe vertigo for eleven days, which disappeared two days after a radical mastoid.

During this vertigo external objects seemed to rotate to the left, and he said he staggered to the left. Was never dizzy in his life previous to this.

About August 12th there was a complete paralysis of the right facial nerve, together with an acute mastoiditis.

He was admitted to my service at the Harlem Hospital on August 18th, with an acute mastoiditis and facial paralysis of the right side. Some hearing was present in the right ear, as determined by eliminating the other ear with the Bárány noise apparatus. There was no nystagmus.

The radical mastoid operation was performed by Dr. Perry Schoonmaker, and the patient left the hospital within ten days.

No signs of fistulæ were discovered at the operation.

He reported for treatment a few times after leaving the hospital and then disappeared.

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*Presented before the New York Academy of Medicine, Section on Otology, April 14, 1916.

On January 24th, about five and one-half months after the acute mastoiditis, he was again admitted to the Harlem Hospital, complaining of some pain and a profuse discharge from the right ear.

On examination a large sequestrum was found lying nearly detached in the external auditory canal. This was removed and the discharge and granulations quickly improved.

The following tests were made: Caloric (cold), right side negative; left side positive. Rotation test to the right, nystagmus fourteen seconds; rotation to the left, nystagmus nine seconds, i. e., stage of compensation. The right cochlear nerve was nonreacting. The left was about normal and there was no reduction in the bone conduction for the C fork.

Description of the Sequestrum.—The greatest diameter of the sequestrum is seventeen millimeters and extends from a point 2.5 millimeters anterior to the canal of the first turn of the cochlea to a point in the nonampullated end of the external semicircular canal, four millimeters posterior to the vestibule.

The greatest width (ten millimeters) extends through the inner wall of the vestibule.

The following principal landmarks can be made out: The internal wall of the ampullated end of the posterior semicircular canal, the end of the nonampullated end of the external semicircular canal, the common end of the posterior and superior semicircular canals, almost the entire inner wall of the vestibule with the fovea hemielliptica and the fovea hemispherica intact, the opening of the vestibular aqueduct, the recess and part of the circumference of the round window, the floor of about two turns of the cochlea with the canal intact for three millimeters in the first turn at the anterior segment. The lamina spiralis is plainly visible in a large part of the first turn. The modiolus is intact nearly to the helicotrema. A large part of the fundus of the internal auditory canal is present, showing the openings for the cochlear and vestibular nerves. The upper internal part of the fundus where the facial nerve should enter the fallopian aqueduct is absent.

The modus operandi of labyrinthine sequestration is interesting, as pointed out by Braun, Friesner, and others.

The labyrinthine blood supply comes almost entirely from the internal auditory artery (arteria acustica centrala Sapolini). which is one of the transverse branches of the basilar artery.

Inasmuch as there is only a slight communication with the tympanic arteries, the internal auditory is pracfically a terminal artery.

When, therefore, inflammation attacks this vessel near the internal auditory meatus, shutting it off, the entire labyrinth dies. This then acts as an irritant to the perilabyrinthine cancellous bone. The resulting inflammation causes granulations to form there, finally producing a separation of the labyrinth en masse.

Another interesting feature was the absence of nystagmus. This is explained by the slow destruction of the vestibular nerve, compensation proceeding step by step with the loss of function of its different parts.

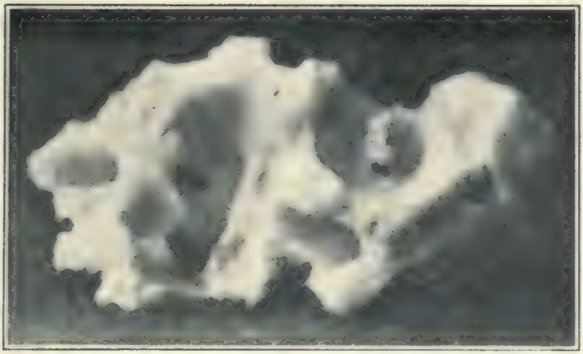

FIGURE 1.

Sequestrum of the labyrinth, external view. Magnified twenty-five diameters.

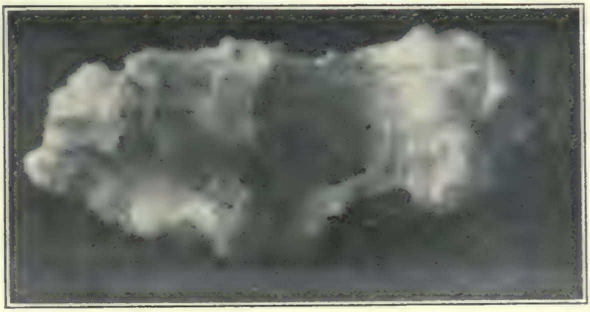

FIGURE 2.

Sequestrum of the labyrinth, internal view. Magnified twenty-five diameters.

LXIV.

ON DIAGNOSIS AND OPERATION OF SINUS THROMBOSIS.*

By J. Holinger, M. D.,

Chicago.

A number of peculiarities of sinus thrombosis will become more evident if we compare this form of general septicopyemia emanating from a well-defined focus with others of a similar kind; for example, with acute otitis media and general infection from disease of the tonsils, nose and pharynx. In acute otitis media the general malaise and fever are evidences of general infection. The focus, namely, the middle ear, inclusive of the cells of the mastoid process, is enclosed in bone, which permits the exchange of blood and lymph to take place only in a very restricted manner through narrow foramina in the bone. This explains, on the one hand, the great pain; on the other hand, the low fever, which hardly ever rises over 102° F., even in extensive suppurations in the mastoid. Embolisms or metastases do not occur.

Secondly, the conditions in general infections from diseases of the nose and tonsils differ in several respects. The parts are well supplied with blood and lymph. Therefore, when they are inflamed we see great swelling, redness and secretion. The nose and nasopharynx are much exposed to infection and apparently have free drainage to the outside. Furthermore, an extensive lymphatic system, namely, Waldeyer's ring, the follicles and the lymph vessels and glands of the neck form strong barriers against general infection. The fever, therefore, is steady but low, 99° to 101° F. Only minutest embolisms and metastases occur. They manifest themselves in rheumatic pains throughout the entire body, especially in various joints and muscles. The infection enters the general system by way of the numerous capillaries and blood vessels from the many folds and pockets of the linings of the nose and throat. Owing to the favorable conditions of drainage of the infectious material to the outside, and also to the smallness of the vessels concerned, the invasion of the general system occurs by means of minutest particles, and often repeated. These particles are so small that they pass the capillary system of the lungs and settle in the capillaries of the joints and muscles.

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*Read before the Chicago Laryngological and Otological Society, May, 1916.

Compare with these two the symptoms as we now know them in sinus thrombosis. The local pain may or may not be present, as there is no retention of secretion. Large embolisms in the lungs are frequent, and so are metastases in the neighborhood of the joints and in the muscles. They often lead to suppurations. The most characteristic symptom is the fever. In the midst of comparative euphoria the patient suddenly becomes restless, excited, feels sick, a chill follows, and the temperature rises like a sky-rocket to 104°, 105°, or even 106° F. In a quarter of an hour, an hour, or so, the whole phenomenon may be passed. These are the typical septic attacks. They are the most characteristic symptoms of sinus thrombosis, and are produced by septic masses being thrown into the general circulation, as pieces of a septic thrombus in the sinus. The masses are retained in the lungs, if they are too large to pass, and form an embolic infarction. If they are smaller they pass the lungs and locate somewhere in the periphery of the body, or are simply overcome by the general resistance of the individual.

The comparison of these two general infections starting from a septic focus in the ear, shows, firstly, in otitis a constant small invasion of the system from a small focus. The export of the septic material from the focus is mechanically interfered with. In sinus thrombosis there is a sudden invasion of large masses of septic material directly into the blood, of short duration. This gives us important differential diagnostic points. In simple otitis there are no high temperatures and no variations; in sinus disease, high temperatures and constant variations.

Now, a sketch of the diagnosis and therapy of sinus thrombosis must be drawn, as it was given in a number of textbooks and works on mastoid surgery in the years 1906-1909 or 1910. The diagnosis was mainly based on the finding of a hard, cord-like swelling along the jugular vein, swelling of the eyelids, septic metastases in the periphery, combined with temperature and chills—all this occurring in connection with middle ear disease. Mastoid symptoms might or might not be present. Swelling and sensitiveness on several typical places in the surroundings of the mastoid, where veins leading to the sinus from outside the skull through the bone, becoming infected from the infected sinus, may indicate the trouble. Oppenheim advised, furthermore, bacteriologic blood examinations. The therapy consisted in exposing the sinus. When in doubt as to the seat of the thrombosis, puncture with the hypodermic syringe, or incision of the sinus were advised. When a thrombus was found, it was followed downward and upward until fluid blood flowed, the hemorrhage stopped by tampon, and the thrombus removed. The ligature of the jugular vein was done either before the operation or during it. This, with a few modifications, is the teaching of the following textbooks and special works on surgery of the mastoid: Whiting, 1905; Körner and Oppenheimer, 1906; Barnhill, 1907; Bing, Politzer, Hartmann, Schwartze-Grunert, Heine and Kopetzky, 1908; Dench, Politzer, Ballin, Heller, Ostmann, West-Scott (London), 1909; Lake, Gray, Urbantschitsch, 1910. Hasslauer, 1911, and Heine, 1913, have more modern standpoints. A few dissenting opinions must be mentioned. They are Bezold's textbook, 1906, and Boeninghaus, 1908, which take issue against ligation of the jugular vein. Dench could never find the cord-like band of the jugular, and objects to exploration of the sinus by puncture. A surprising statement is in Politzer's textbook, 1908, page 523. He says: "Chills, high fever, even pain along the jugular vein, do not absolutely justify the supposition of the presence of a sinus thrombosis. Even cases like the one which Prescott-Hewett[1] describes as cured, where, besides chills, metastases in the lungs and joints were also found, must not unconditionally be considered as cured cases of sinus thrombosis, since a similar complex of symptoms may occur without sinus thrombosis, through absorption of infectious material from the osteophlebitic spaces of the temporal bone." A later edition of this book does not contain this paragraph. Not less odd is the statement in Gray's textbook, saying that the author has no experience of his own about different operations on the bulb of the jugular vein.

Strong objections against these teachings are apparent at first sight. No differentiation between acute and chronic cases was made. Cord-like swelling of the jugular vein, swelling of the eyelids, could only be symptoms of very late, practically final, stages of the disease. The first one shows that the thrombophlebitis has progressed to within a few centimeters of the heart, and the swelling of the eyelids is evidence that the thrombosis has spread over a great part of the blood sinuses at the base of the brain. The objections against the modus operandi are equally strong. Koerner showed that the mortality after ligature of the jugular vein is not less than without it. A number of points may be cited which make it evident that in some cases death may be and really was due to ligature. In the first place, it certainly increases the shock of the operation; in the second, all the veins which enter the jugular from the base of the skull to the ligature, a distance of from five to eight centimeters, will become septically thrombosed; in the third, the handling of the jugular and the ligature may loosen crumbs of the thrombus, and throw them into the general circulation, the very thing we try to avoid; and, in the fourth, Nürnberg[2] cites a case of death from arrosion and rupture of the carotid artery, due to the ligature material of the jugular vein. None of these dangers or accidents can be minimized or avoided. In Scheibe's clinic, Schneider[3] wrote a paper: "Should the Jugular Vein be Ligated in Sinus Phlebitis Caused by Acute Purulent Otitis Media?" He had, without ligature, one death in thirteen operated cases, 92.03 per cent recoveries. He draws the conclusion that the ligature of the jugular vein is superfluous and worthless in almost all cases of acute suppuration. Literature shows that in many cases it has done harm. The best advice is to confine the ligature to those rare cases where the jugular vein itself is diseased.

Our operative procedures on the sinus must be influenced by the conclusions drawn by Heymann[4] in his "Experiments on Sinus Thrombosis and Otogenous Pyemia." He says: "(1) Infectious sinus thrombosis can best be produced by laying an infected tampon against the external wall of the sinus. Simple painting or rubbing of emulsions of bacteria on the wall of the sinus, even after superficial lesions were produced, does not usually cause coagulation. Sterile compression of the sinus does not always cause coagulation. (2) Germs may enter the blood stream without the means of a thrombus. A thrombus at the place of infection is not a necessary condition for the occurrence of autogenous pyemia. (3) A sinus thrombosis usually begins as a thin parietal coagulum, which may grow to become obstructive. A large part of the blood in the sinus may coagulate so quickly that it causes the impression of coagulating all over at the same time. (3-a) The time from the infection to the coagulation varies greatly, as does the time from the first coagulation to the formation of an obstructing thrombus. (4) The thrombi may contain germs from the beginning, but may also become infected later on. The germs usually enter the thrombus through the wall, but they may also be caught from the blood current. (4-a) The number of bacteria in the thrombus varies greatly. It is usually less at the ends. A moderate number of bacteria does not interfere with organization of the thrombus. (5) A thrombus may remain adherent and nonobstructing, and become organized. (6) The growth of a thrombus usually progresses from the center. Intermittent thrombi often are produced from thrombosed veins of the dura which cause new thrombi after the first one has been absorbed. (7) Parietal thrombi have a strong tendency to spontaneous healing; even malignant thrombi heal. The infection often causes destruction of the wall; even recanalization. (8) The macroscopic appearance of the wall of the sinus does not allow conclusions as to the contents. (9) In bacteremia an aseptic thrombus becomes infected at once."

Equally important are the conclusions which Streit[5] derived in his paper, "Further Contributions to Histology and Pathology of Meningitis and Sinus Thrombosis." He says: "A thrombus usually starts from the wall of the sinus as a consequence of inflammatory changes and infection of the wall of the sinus. This first and most important cause is, however, not sufficient. The infiltration of the wall may reach such a degree that it almost closes the sinus. Yet no thrombus will form as long as the endothelium is intact. Predisposing causes for the formation of thrombi are changes of the normal blood current, like the formation of whorls in strictures or local enlargements of the sinus."

To these experimental results we must add some observations where, after operations, thrombi formed in the previously normal sinus at places where exploratory punctures or incisions had been made, and after unintentional injuries or even after simple exposure of the normal sinus (Bondy[6]: "Sinus Thrombosis After Exposure of the Normal Sinus").

Finally, attention must be drawn to the descriptions in Bezold's textbook of large flat mastoid cells along the sinus, with extremely thin bony walls, or even dehiscences. In such cases every acute otitis media is practically an extradural abscess around the sinus.

There is no need of any further comment on these arguments. The experimenters agree that sinus thrombosis has a very great tendency toward healing, if we only succeed in keeping reinfections (perisinual accumulations of pus) away. The blood current will get rid of the thrombus, sterilize, organize and absorb it, if we only take care that the endothelium is not lacerated.

The following conclusions may be drawn: The diagnosis of sinus thrombosis is made from septic attacks. We can observe four to five attacks of chills and high temperature, in order to ascertain the diagnosis, without danger to the patient. It is not necessary to operate after the first or even after the second attack. Other sources must, of course, be excluded. The comparison at the beginning of this paper shows that in uncomplicated inflammation of the middle ear, even when the mastoid cells are extensively involved, the temperature does not rise above 102° F. As soon as we have temperatures of 104° to 105° F. in acute or chronic inflammations, we know that the inflammatory process is not confined to the middle ear. Every acute otitis media must be watched for septic attacks. It is a good plan to verify the statement of the patient about the presence of chills and high fever, either at a hospital or by a competent nurse. Other mastoid symptoms may or may not be present.

The operation consists in laying the sinus bare very extensively. Handling the vessel, palpation, incision, puncture, are to be avoided, because they are bound to cause injury to the endothelium. As a consequence, new thrombosis sets in and interferes with the organization and absorption of the old ones. Splitting the sinus and removing the thrombus till the blood flows from either end force you to tampon again and cause new thrombi. In accidental injury to the sinus, tampon lightly, so as not to interfere too much with the blood stream. Ligation of the jugular vein is reserved for very old cases with extensive metastatic abscesses.

A few cases may be mentioned briefly:

Case 1. Miss H., eighteen years old, daughter of a colleague, had acute otitis and several septic attacks. At one of my visits her mother cautioned me of an oncoming attack. The patient became very restless, complained of headache, chills developed, and the temperature went up to 104½° within seven minutes. After a few more minutes she became quiet again, and after twenty minutes everything was over. The next day Dr. Morgenthau and I operated. An abscess around the sinus was drained, but the sinus itself was left untouched. She made an uninterrupted recovery. This was ten years ago. Since then she has never had any symptoms that may be traced to sinus thrombosis.

Case 2. Ten days after the operation of a boy of six, where an extradural abscess was drained, the boy played about the room, when suddenly he stopped and cried out for pain in his right knee. The next day the knee was hot, swollen and extremely painful. Rest for a few days brought complete recovery. A septic embolism was overcome without any further disadvantage.

The enumeration of more uncomplicated cases is unnecessary.

This spring four cases were operated. Three of them showed points of special interest.

Case 3. December 29, 1915, G., girl three years old, referred by Dr. McNeill. Had a cold and suppuration from both middle ears since Thanksgiving day. During the last few days, repeated chills and temperatures up to 105½° F. set in. Mucopurulent discharge flowed from the nose and ears. The discharge from the left ear was fetid. The perforation was behind the short process. Hearing: Loud conversation. All other organs were normal. Operation of both mastoids, December 31. An extradural abscess was evacuated on the right side. The sinus was exposed down to the vicinity of the bulb. On the left side some decayed bone was removed. During the first week in January several temperatures, up to 104° F., were recorded. The child left the hospital January 31. Both mastoid wounds were closed. February 29, both perforations are closed; hearing of whispered voice at more than seven meters distance.

Case 4. K., child fifteen months old, referred by Dr. Schott. Had just gone through an extensive cleft palate operation by Dr. Brophy; then pneumonia and suppuration of right ear. For the last few days she has had irregular temperatures, up to 104° F. The perforation is in the lower posterior quadrant· the region behind the ear is sensitive on pressure. An observation of several days showed that the child was getting rapidly worse. Operation June 12, 1916. The posterior wall of the antrum is formed by the sinus, which is bathed in pus. At the subsequent dressings the wound was syringed with warm boric acid solution. The temperatures kept up for another week, then gradually dropped, and the child was discharged July 3, 1916, with perforation closed, and, as far as could be ascertained, normal hearing.

Case 5. H., boy four years old. Referred by Dr. Kerlin, on account of suppuration from left middle ear, on May 27, 1916. There was also hardness of hearing, and high temperatures had been recorded. Perforation in posterior upper quadrant. The boy played all day after a few treatments. May 31 the suppuration stopped, the perforation closed and hearing became nearly normal. At 4:00 p. m. temperature was 104° F. For the next few days short periods of high temperatures showed nearly every day. No sensitiveness on pressure behind the ears. Operation, June 5, at St. Joseph's Hospital. The antrum, aditus ad antrum and a few small cells were normal. On the rear margin of the wound of the bone, on a level with the floor of the antrum, a tiny drop of yellow pus forced itself out of a pin-point opening of foramen nutritium. An extradural abscess containing a teaspoonful of pus surrounded a completely necrotic sinus to the extent of an inch and a half. The loose necrotic parts were removed with forceps and scissors; the thrombus itself was left intact. During the next few days a few high temperatures were registered, but healthy granulations rapidly covered the necrotic area. Bacillus mucocus capsulatus was found in the pus. June 20 the boy went home. June 30 the wound healed; hearing and drum membrane normal.

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LXV.

A STUDY OF ONE HUNDRED CASES OF MASTOIDITIS AND ITS COMPLICATIONS REQUIRING OPERATION.*

By Jesse W. Downey, Jr., M. D.,

Baltimore.

A recent study of my case histories in one hundred consecutive operations for mastoiditis and its complications has been of such value to me that I trust a short review of these cases may be of some general interest. I shall give the statistics as briefly as possible and shall omit details of case history.

There were ninety-two patients in this series of cases, the youngest being ten weeks of age and the oldest seventy-five years. Forty-six per cent of the cases were in children under ten years old. The mortality was ten per cent; the complications causing deaths being as follows: Septic meningitis, three cases; cerebral abscess, two cases; cerebral tuberculosis, three cases; general septicemia (scarlet fever), one case; lobar pneumonia, one case. Two of the cases died six months or more after operation, but the mastoid wounds had never healed. In one of these cases in the service of a colleague† at another hospital, the tuberculosis of the brain was certified by autopsy. The other cases, from clinical signs, probably died from the same condition.

A few moments' consideration of the mortality in these cases is of some importance. Two of the patients died of general septic conditions, one being in the acute stage of scarlet fever at the time of operation. The other developed a lobar pneumonia one day after operation. Three of the cases were undoubtedly tubercular, one certified by autopsy; but whether or not the origin of the tuberculous process was aural, it is difficult to say. I am inclined to think, however, that it was, as all three patients were negro children, two being one year of age, the third, seven years old; each had had a discharge from the ear varying from six months to three years in duration. All were apparently hopeless cases from the first.

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*Read before the Medical and Chirurgical Faculty of Maryland at its annual meeting, April 27, 1916.

†Dr. C. A. Clapp, Maryland General Hospital, the patient having left the clinic at the Baltimore Eye, Ear and Throat Hospital.

Of the five patients who died from strictly otitic complications, but one of them had had any medical attention before serious symptoms arose. This case, I think, I shall remember as long as I practice otology, for, regardless of the most careful treatment and with the advice of two otologists of much greater experience to guide me, the patient went successively through the stages of an otitis media, mastoiditis, cerebral abscess to a most terrible death from meningitis. Three cases dying of meningitis had had discharging ears in two instances of only six weeks' duration; the other one had had an otorrhea all his short life of six years, and none of them were brought for treatment until alarming symptoms had arisen.

In the last few years most brilliant work has been done seeking to cure by drainage septic meningitis and cerebral abscesses of otitic origin. The results have been more promising in the latter than in the former class of cases, but at the best, the attempt to save these patients, once the complication has arisen, is usually futile. If, however, we compare the high death rate in the mastoid surgery of thirty-five or forty years ago, when the operation was undertaken only in the advent of grave symptoms, with the low mortality of the present day, the prevention of the more serious complications becomes evident and is obviously the result of early mastoidectomy, indicated by definite symptoms of an infection localized and confined to the mastoid process itself.

SYMPTOMATOLOGY AND DIAGNOSIS.

A discharging ear is always a menace. When of long duration, every effort should be made to cure it by appropriate and conservative treatment, but when such means fail and there are granulations in the middle ear or polyps extending into the external auditory canal, or if the patient complains of pain, headaches, vertigo and nausea, a radical mastoid operation should be advised. It is quite true that a person may have a discharging ear for years without ill effect; it is equally true that the same person may die within a few days from an acute exacerbation with an extension of the infection to the meninges or the brain.

An acute purulent otitis media will yield quickly to treatment in at least seventy-five per cent of the cases.*

Primarily, the symptoms of acute mastoiditis are a discharging ear, tenderness on pressure over the mastoid antrum and tip, and fever. All these symptoms may be present in the very early stages of an acute infection of the middle ear, especially in children, to clear up immediately following a wide incision of the drum head and the establishment of free drainage. It is the persistence of any or all of these symptoms which is important.

Just here, for the sake of brevity, I shall make certain sweeping statements which I know are open to discussion, but which constitute safe rules to follow. First, there should be no mastoid tenderness or fever after three days of free discharge in a case of acute suppuration of the middle ear. Secondly, the case should yield quickly to treatment and the discharge should cease within ten days. The persistence of symptoms beyond this time does not necessarily indicate the need for an immediate mastoidectomy, but their continuation undoubtedly means an infection of the mastoid cells. In naming the primary symptoms of acute mastoiditis, I have purposely omitted mentioning swelling with fluctuation over the mastoid process. This is seldom an early sign, even in children, in whom it is more often seen than in adults. As it indicates a perforation of the cortex and a subperiosteal collection of pus, it rarely occurs earlier than three weeks, and usually about six weeks after the beginning of the infection.

Certain characteristics of the major symptoms are important to note. A persistent, pulsating discharge which is profuse, in spite of frequent cleansing of the external auditory canal, indicates that the mastoid antrum is involved; sagging of the posterior wall of the external auditory canal accompanying such a discharge may be considered pathognomonic. Fever some time during the day is almost always present. Much could be written on the temperature course in these cases. I wish to emphasize but one point at this time, and that is that after the initial rise (of an acute purulent otitis media), which may be considerable, the temperature is seldom high in an uncomplicated case of mastoiditis after drainage from the middle ear is established; therefore, the temperature course must be carefully watched, and a rise to 100° F. or even 99° F. must be considered as fever, if it occurs some time during each twenty-four hours.

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*One hundred and twenty-five patients were treated at the Baltimore Eye, Ear and Throat Hospital from October 1, 1914, to October 1, 1915, for acute suppurative otitis media; thirty cases were operated on for acute mastoiditis.

Tenderness over the mastoid, especially over the tip, is not uncommon in the first forty-eight hours of a severe middle ear infection, and indicates intense congestion of the bone. It should clear up rapidly after the middle ear is draining. If it persists longer than three days and becomes localized over the antrum, increasing from day to day, it takes on pathologic significance. Once middle ear drainage has become established, mastoiditis is not usually a painful condition, except in the intensely congestive forms. The patient complains chiefly of a sense of fullness in the mastoid region and of a throbbing sensation in the ear.

It is impracticable to summarize, except in a general way, diagnostic signs or the indications for operation in this series of cases. The three cardinal symptoms I have mentioned were often so well marked that the diagnosis was a very simple matter. On the other hand, there were a number of vague cases in which laboratory examinations proved of marked service. A differential count of the white blood cells was of much value in some obscure cases, increase of the polymorphonuclear cells indicating pus. Even a simple count has been of marked service. One should not expect a high leucocytosis, but a count of from ten to twelve thousand is suggestive of an involvement of the mastoid cells; and in many cases in which the count has not been above this, I have found pus in the mastoid cells. I wish to particularly emphasize the observation that the high leucocytosis of general infections will not be found in an uncomplicated mastoiditis, but the moderate increase in the number of cells should not be disregarded.

How soon after the middle ear infection may the mastoid operation be indicated? In this series of cases I have opened the mastoid one day after earache was complained of, and without discharge from the ear, and found pus in the mastoid cells. I do not believe, however, that the history was correctly given in this case; therefore, the infection was probably of much longer duration.

I have several times in this paper called attention to marked symptoms of mastoiditis accompanying the very early stages of an acute purulent otitis media which clear up almost immediately once free drainage from the middle ear has been established. A few of these patients, however, continue to exhibit alarming symptoms for from one to three days after a free myringotomy, and the surgeon is justified in opening the mastoid, especially if there are signs of meningeal irritation; on the other hand, discretion is often the better part of valor, and if there is free drainage, a wait of from twenty-four to forty-eight hours will often show an abeyance of the fever and tenderness. If after three days the symptoms are still marked, then discretion, socalled, may become a matter of taking chances. In these very acute cases, when they come to operation, one must not expect to find quantities of pus or cellular destruction—intense congestion of the bone may be all that is present, and the operator may be surprised when he finds that the toxic symptoms often persist for several days after the mastoid process has been fully opened and is draining, and that the healing is always slow. Pathologically such cases are primarily congestive, due to nonpus forming microorganisms, and their rapid recovery depends upon the general resistance of the patient to the infection. A safe rule to remember is that the recovery must be as rapid as the symptoms have been alarming, and that the danger of complications increases as the symptoms persist, for undoubtedly this is the most dangerous type of mastoiditis. As a rule, the operation is rarely indicated under a week following an acute purulent otitis media, and the average time is from two to three weeks.

Patients with chronic discharging ears may, of course, have an acute exacerbation demanding operation at once. In my series there were eight cases of this character, with a mortality of forty-four per cent. It is a most striking example of the seriousness of chronic suppurative otitis media when it loses its chronicity to flare into activity. In contrast, there were in the series nine patients upon whom the radical operation was performed to cure a chronic suppuration while it was yet quiescent. There were no fatal cases, and the results were satisfactory.

I wish to especially speak of the symptoms in infants and young children. Tenderness over the bone is often difficult to correctly elicit, but slight edema over the mastoid, extending above the ear, is more frequently seen than in adults, and slight enlargement of the preauricular glands and the superficial cervical glands is often present, and is a reliable indication of involvement of the mastoid cells. The temperature is usually higher and the otorrhea more marked than in adults. Also, the leucocyte count is usually higher than in adults.

I have often felt that an otorrhea continuing longer than six weeks, even though there be no other symptoms, and which is not dependent upon an inflammatory condition of the nasopharynx, is sufficient indication in children under five years of age for a simple mastoid operation or the modified radical operation. I have not, however, had the courage of my convictions sufficiently strong to urge the procedure unless there were other indications.

THE SURGICAL PROCEDURES.

The operations for mastoiditis and its complications required in this group of ninety-two patients numbered one hundred and fourteen, which may be classified as follows:

	Cases
(1) The simple mastoidectomy for acute mastoiditis	70
(2) The simple mastoidectomy for acute recurrent mastoiditis	9
(3) The secondary mastoidectomy to promote healing	8
(4) The mastoidotympanal exenteration, or the socalled radical operation, indicated by acute symptoms	8
(5) The radical operation, indicated to cure chronic suppuration	9
(6) The modified radical, or socalled Heath operation	4

(7) The ligation of the internal jugular and obliteration of the lateral sinus	5
(8) Subtemporal decompression	3
Total	116*

THE OPERATION FOR ACUTE MASTOIDITIS.

It is not the purpose of this paper to go into the operative technic. It will be sufficient to say that the simple operations consist in freely opening the mastoid process into the antrum and the removal of all necrotic bone. The anatomy of the process varies greatly, from the large cellular type to the diploetic type with dense cortex and small cells. The former type of mastoid is more often found extensively necrosed and filled with pus, even though the symptoms may have been particularly mild. The patients with dense bone formation are apt to complain of much pain on pressure, the temperature is apt to be higher, and on operation the bone is found extremely congested, bleeding at every touch, but with only a small amount of pus present. The infecting organism is, of course, responsible for the pathologic condition found. With the free pus forming bacteria, such as one of the staphylococcic groups, the destruction may be extensive with mild symptoms and rapid recovery. The cases caused by the streptococcus mucosus, for example, may show very mild symptoms and little destruction, and yet go on to serious complications. The cases following grip are apt to be extremely rapid in onset, severe and slow in healing.

The anatomic formation found on operation should be the guide as to the extent of the operation. The cellular structure should be completely opened in all cases, especially down into the tip and back over the lateral sinus. Children over two years of age, as a rule, present fully developed tip cells, and often surprisingly large mastoid processes with cells extending back into the occipital bone and forward into the zygoma.

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*Two cases had recurrent mastoiditis, and at the second operation the modified radical was done; these cases should, therefore, count as two instead of four operations. Corrected total 114. Also, as several of the operations were simply recurettements under nitrous oxid to promote healing, I have chosen to consider the series as consisting of one hundred operations.

If the dura is found exposed, or is exposed at operation, it should be let alone and no granulations adherent to it removed.

Two methods of completing the operation are in vogue. One is to clean out the bone, pack with gauze and leave the wound completely open. The other is the blood clot method devised by Blake of Boston and so ably developed by Reik of Baltimore. In this method the mastoid cells are cleaned out until all diseased portions are completely removed; the wound is allowed to fill with blood, and the skin incision is sewed up completely, with the hope that it will heal by first intention. In the first method the quickest time in which complete healing may be expected is six weeks, with a resultant depressed scar. In the second method healing is complete in one week, and there is a hair-line scar. Before speaking of these methods in detail, I wish to emphasize that my experience has been that there is no midway method—either leave the wound open or close it completely. Having had the privilege of assisting Dr. Reik and seeing his results in many of his blood clot operations, and from my own experience, I am willing to unqualifiedly say that the method is ideal, whether or not primary healing is obtained. Ideal, because it demands a painstaking operation which is of advantage, even when healing by first intention is not obtained. The opponents of the operation recount their failures and damn with faint praise their successes. We praise our successes, even when few in number, and count these few worth the numerous trials to achieve perfection. That the operation is safe, may be deduced from this fact, that whereas many have criticised, no one has yet reported a serious complication directly arising from its use.

In my series of cases I have sewed up completely in forty-five cases of acute infection. I wish it distinctly understood that these cases were all acute, and I do not include the radical operations, the modified radicals, or the recurrettements to promote healing.

In the forty-five cases that were treated by the blood clot method, I had fourteen cases, or thirty-one per cent, to heal absolutely by first intention. Ten cases broke down partially, but the healing was complete in from one to four weeks.

Twenty-one cases broke down completely and required the usual six weeks to two months of dressings before union was complete. I feel that any method which will increase my own efficiency thirty per cent and save one-third of my patients weeks of inconvenience, is a method worth all the time one may spend on it.

I have undertaken to analyze these cases, for it is out of all reason to expect healing in every case of the acute infectious nature of mastoiditis, but many of my deductions are too theoretic for publication at this time. I believe, however, that it would be possible by a careful study of certain symptoms and factors in a large number of cases to select certain cases best suited to the method. Such a study must take into consideration the duration of the disease, the infecting organism, and the general resistance of the patient. It is not the very early case that heals by first intention, but the case of from three to six weeks' duration, in which there is free pus and a breaking down of the mastoid cells.

Of the fourteen cases healing by first intention, twelve, or eighty-six per cent, had an infection of the middle ear, and probably of the mastoid cells, of over three weeks' duration; whereas, in the cases which failed to heal, seventy-one per cent were operated on in less than three weeks after the initial infection. A moment's consideration of the pathologic finding in these cases will show that this deduction is not entirely a theoretic assumption. In the early stages of a mastoid infection there is, of course, an intense congestion of the bone—the cells are not broken down, but are found filled with a serous exudate. Even after the complete removal of the cellular structure, the inner table of bone will be found markedly congested; in other words, there is as yet no line of demarcation between the healthy and the diseased tissue. Both local and general resistance is insufficiently established at this stage. In the later stage of the infection we find much less congestion, the cells are broken down, and there is an abundance of free pus, granulations and necrotic tissue, which, when cleaned away, leave a white nonbleeding inner table of bone. The general resistance has been established, and we are, therefore, much more likely to get rapid healing.

To gain rapid healing in a large percentage of cases it would be better perhaps to wait until the acute inflammatory symptoms had subsided, and I think we all are averse to operating too early, not solely on this account, but because the reaction following an early operation is apt to be more severe; on the other hand, when definite and severe symptoms of mastoid involvement appear very early, we are often forced to operate for fear of further extension and resultant complications over which we have so little control, and early drainage of the mastoid cells is by far the safer course to follow.

Concerning the secondary mastoid operation to promote healing there is little to say. Several of these were but recurettements under nitrous oxid; but as a rule the mastoid cavity was entirely reopened and cleaned of granulations and then sutured. In these cases it was almost universal to find some necrotic bone and unhealthy soft granulations. The results, as a whole, were satisfactory, and healing was hastened.

The operations for infectious recurrence present little of interest. It is not uncommon in children to have an old mastoid wound break down during a reinfection of the middle ear, even years after the first operation. The cellular structure of the bone does not reform; but it is not unusual to find a reformation of the cortex almost complete, if there has been sufficient time between the infections.

I have spoken elsewhere of the seriousness of the chronic suppurative ears. This has been most forcibly brought home to me by the high mortality in the cases upon which the radical operation was indicated by an acute exacerbation. The cases upon which the radical operation and the modified radical were performed to cure a chonic suppuration are too few in number to furnish observations of any value.

THE COMPLICATIONS OF MASTOIDITIS.

In this series of cases there were five patients who developed thrombosis of the lateral sinus. The internal jugular vein was ligated, the lateral sinus exposed, opened and obliterated. All the patients recovered. The number of cases is small, but all were of great interest, and certainly of value from the standpoint of diagnosis.

The diagnosis of the conditions is by no means as simple as the textbooks would have one believe, for the reason that the symptoms in the early stage are seldom so severe as those described. In a case which has progressed to the stage of an organized thrombus, chills, sweats and great prostrations are often marked. When the condition has not advanced beyond a state of phlebitis, the temperature course is, as a rule, the only sign of trouble. Of course, the severity of the infection determines the rapidity of the stages between a phlebitis and a complete obstruction of the lateral sinus, but the earlier the diagnosis the greater is the patient's chance for recovery. In only one of the cases was a severe chill the initial symptom, and this man, although he was operated on within a few hours, later developed metastatic abscesses. One or two of the cases ran a septic temperature for a week before operation, and yet made perfect recoveries without complications. I believe the temperature course to be the most valuable diagnostic sign. An up and down fever course, evening rises to 101° to 102° F. or more, with morning remission to 99° F. (seldom, if ever, does the temperature drop to normal), is almost pathognomonic. There may be no chills and the patient may feel "fine," but he will take on a septic look in a day or two. Also, I most thoroughly believe in the value of laboratory findings in these cases. If a phlebitis exists the leucocytes will be increased to 18,000 to 20,000, with a relative increase in the polymorphonuclear cells. In two cases with an almost typical temperature course, I have deferred operations because the leucocyte count was normal—seven to eight thousand—to find later that in one case[1] the temperature was due to an obscure visceral luetic lesion, and in the other case to a tubercular focus in the lungs. The blood culture work of Libman and Cellar is of value, but here so much time is lost unless the cultures grow quickly that the examination is robbed of some of its value. A positive result obtained quickly is confirmatory. The ophthalmoscopic sign mentioned by Crowe some years ago has not proven of value; to be positive there must be a complete obstruction in the lateral sinus, and this is not present in early cases. That the venous obstruction may be seriously affected, however, is certain, and that this may be seen after a ligation of the internal jugular has been proven in one of my cases, in whom marked choked discs developed after operation. This case[2] was reported in full by Dr. Harry Friedenwald and myself before the American Otological Society last summer. Subtemporal decompression gave immediate and permanent relief.

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[1] Downey: Symptoms of Thrombosis of the Lateral Sinus. Report of a case in which an exacerbation of visceral lues simulated the symptoms of the lateral sinus thrombosis. Annals of Otology, Rhinology and Laryngology, Vol. 21, No. 4, p. 1044.

One case developed a cerebellar abscess, which was drained, and the child made a perfect recovery. I am indebted to Dr. Charles Bagley for this cerebellar operation and for two of the decompression operations.

Before leaving the subject of complications, I wish to say that the cases of cerebral abscesses,[3] all of which died, have been of great concern and interest to me. These cases are nearly all without symptoms of localization, and are very vague up to a point when all help is futile. There is one feature, however, that I wish to mention, and that is that persistent headache following a mastoid operation or accompanying a chronic suppurative otitis media is always a grave symptom, and should be given the deepest consideration. Some day, when we learn to diagnose cerebral abscess in an early stage, and have the courage to operate with only those signs which now seem insufficient, we are going to be able to save a creditable percentage of these patients. Headache has been the only symptom in every case I have seen, until the very last stages.

In my summing up in this series of cases, if my conclusion seems dogmatic and based on an insufficient number of patients, may I say that much has been drawn from the writings of others to be proven by these cases, and, at least, they serve as the basis for further observation and study.

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[2] Friedenwald and Downey: Decompression for the Relief of Choked Disc Following Thrombosis of the Lateral Sinus. Annals of Otology, Rhinology and Laryngology, Vol. 24, No. 3, p. 613. Transactions of the American Otological Society, Vol. 13, Part 3, p. 503.

[3] Two cases, not included in this paper, admitted to my service at the Baltimore Eye, Ear and Throat Hospital for observation; complained only of headache. They both had chronic suppurative otitis media. Both suddenly collapsed and died in coma before the mastoid operation was performed. Autopsy in one case, located an abscess of the temporal lobe.

LXVI.

THE UPPER RESPIRATORY MUCOUS MEMBRANE AS A KEY TO ERROR IN METABOLISM.

By CHARLES WALDO STICKLE, M. D.,

BROOKLYN.

The object of this paper is a plea for more rigid investigation of the primary causes of upper respiratory obstruction not due to deformities, infective processes or new growths.

The mere fact that a nose does not afford sufficient space for unobstructed breathing seems to warrant, in great plenty, for the demand that something must be removed from that organ; if not something to be removed, that it needs local treatment of some sort or another. This also applies to any of the abnormal conditions met with in the pharynx and larynx not due to local disease or deformities.

There is a long educational road and many a disheartening experience before the patient investigator who will steadfastly endeavor to make clear to the family physician and to the sufferer that general disturbances of any type whatsoever first disturb the equilibrium of the circulation and thereby place its unfailing indicator in the mucous membrane of the upper respiratory tract, as a venous stasis from peripheral nerve ending paralysis, resulting in dilatation of the peripheral blood vessels with its accompanying relaxation of the mucosa.

So striking are the ramifications of the mucous membrane index in connection with the manifold ailments of the body, so absolutely true is the pointing of the hand, it is to be regretted that it is a hidden factor for alarm to so many of our general physicians.

In this class we have to deal with more than eighty per cent of all nasal cases and, by its influence, in a great majority of inflammatory otitic, pharyngeal and laryngeal cases. In none of this eighty per cent of nasal obstruction is the removal of any portion of the mucous membrane justifiable, unless it is found that the posterior turbinate ends are degenerated from long standing engorgement, with a resulting chronic hypertrophy or hyperplasia, of itself obstructive, or the same condition in the body of the turbinate, and then only to remove the hyperplastic bone or soft tissue with as little disturbance of the mucous membrane as possible; nor is it admissible, except in hyperplastic cases, to use the actual cautery or the various escharotics in everyday use.

Every scar or injury to the natural outline or surface of the mucosa will, in health, make its presence felt. None, absolutely none, of the everyday nasal washes or commercial nasal applications, which are numberless, are of any value whatsoever, whether used in spray, douche, snuffing, etc. The actual result is to only diminish the protective value of natural secretions in each and every case, to say nothing of acting as mechanical carriers of infective processes to surrounding areas. In other words, plainly stated, no local treatment whatsoever is indicated. The arts of medicine must be employed to meet the personal demands of the sufferer.

In actual practice, arriving at the end results by their delay, it must be admitted, is apt to be rather discouraging. The whole subject is one of detail attention to the physiologic chemistry of metabolism.

When it was above stated that the effort of this paper was to apply only to the obstructions due to noninflammatory causes, it was not meant to convey the idea that this venous obstruction was not present in the inflammatory, infective or deformed cases. On the contrary, it is rarely absent, simply causing additional obstructions and, in a great many cases, a cause for disappointment in the results of surgical procedure; such as continued hoarseness after complete healing of an operated larynx; continued suppuration and engorgement after the exenteration of an ethmoidal labyrinth: a still greater obstruction after the resection of a deviated septum or a continuance of an otitic mucous inflammation. You have your cause, without any fear of contradiction, either in some interference with metabolism, whether it is from an organic disease, an infective disease, a disease of the gastrointestinal tract, or from a parenteral anaphylaxis. There is but one word to use as the positive starting point in any one or all of these conditions. That word is protein.

It is at present our good fortune to have in this country, as well as in foreign countries, several men, eminent physiologic chemists, who have for many years devoted themselves to a train of thought and to chemical experiments, the basis of which is the poisoning from protein or from its diverse chemical combinations. These men have shown us the process wherein the body becomes prey to itself through its inability to create an immunity to each poisonous protein derivative, each in itself producing its particular sensitization, and also how, by the absorption of the poisonous products of gastrointestinal putrefaction or fermentation, the functional destruction of the body cell or its disease change is accomplished.

They will eventually discover substances inhibitory to the activity of foreign protein poisoning by creating a field within which these poisonous products will not develop, or within which they will not be absorbed, or, if absorbed, will be immediately counteracted or destroyed.

The efforts of the writer have in the past several years been particularly and interestedly directed, in this class of conditions, toward the relief of the local symptoms due to general causes, considering particularly the end results governing only the cases of obstruction due to simple blood engorgement—i. e., infiltration from venous stasis.

It is not my purpose to go into an analysis of the various anaphylactic or toxic changes from any particular protein by-product poisoning, whether bacterial, animal or vegetable, whether enteral or parenteral, or as to the conditions which cause the various body cells to become so loaded with these products as to become indefensible through ensuing inability to produce their various ferments for the assimilation of these protein poisons, or to be functionally destroyed by the various toxin byproducts of putrefaction or fermentation.

The physiologist tells us why when a human has yellow fever and recovers, he is then immune to the disease; but, when the sensitization from a foreign albumin has occurred, that subsequent exposures increase the susceptibility.

He has shown us how death suddenly occurs by paralysis of inhibitory nerves of the heart from absorption of toxins in great numbers.

The physiologic picture is the same in the class of cases to which I wish to call your attention, but, of course, in an attenuated degree. The same exposures, the same susceptibility, the same absorption, the same engorgement, dilatation and relaxation exemplify the simple cases of mucous membrane infiltration or turgescence, of which there are so many and by which we are so pressed and tempted to resort to surgical measures.

The physiologist has not told us how we can clinically differentiate in all cases between the action of the anaphylactic poison products and the toxic effect of the various putrefactive or fermentative changes which attack the body cells. When he discovers the exact atomic changes, then we will have our exact etiology. Let us hope the time is not far distant; but, in the meantime, let us give thanks for the far-reaching discoveries of the present.

When the rhinologist sees a nasal mucous membrane which is engorged and turgescent, relaxed, infiltrated, insufficient breathing space and, with the means at his disposal, he produces a more or less normal position by driving the blood therefrom; finds a nasal cavity which contains no malformation or new growth, no suppurative process or adjoining disease, an air space sufficient for the needs of the patient, he should know that the elimination is at fault. When the laryngologist sees the fauces, pharynx or larynx infiltrated under same conditions as above stated, he should know the elimination is at fault.

This fault of the circulation is where the mucous membrane reigns supreme as an index. Just as long as it is not normal in position, color or secretory power, just so long one may be assured that the etiologic factor is still in evidence.

From desultory reading of the investigations of the Vaughans, Rosenow, Billings, von Pirquet, Weichardt, Richet and others, and observations in many of these cases, taking into consideration what diverse etiologic factors have been discovered during treatment of these cases, I am inclined to believe that we have to deal with the combined anaphylactic and toxic influence of the protein molecule. Anaphylactic through the exposed mucous membranes or the skin; toxic through the teeth or alveolar, suppurative, inflammatory or organic lesions.

or through the gastrointestinal tract, both processes making their effect objective through the peripheral circulation.

As to whether it is a parenteral heterogenous albumin or a fermentation or putrefaction in the gastrointestinal tract, to all intents and purposes the same objective symptoms appear.

The toxic element from a gastrointestinal, fermentation or putrefaction standpoint has been the factor to which my attention has been directed more particularly. By careful inquiry into the general symptoms, methods of eating and modes of living, one can almost always find cause for disturbance of the general equilibrium. If not, the physical and chemical examination, the microscope, the X-ray, will in all probability clear up the situation.

It is not an unusual thing to have this index point the way for a diagnosis of nephritis or appendicitis, pulmonary tuberculosis or a genitourinary involvement.

A common thing for two or three quarts of cream and milk per day in a two-year-old child to claim relationship to an obstructed nose from which its neighboring mass of adenoid tissue has been removed for the relief of the mouth breathing.

In conclusion, I present the histories of three interesting cases, particularly striking in the results obtained through investigation of the general conditions, which presented in each case the same nasal symptoms from engorgement, infiltration and relaxation of the mucous membranes—retracting temporarily to normal under use of vasomotor stimulation—in which there was found no local cause for the peripheral stasis.

Case 1.—E. T. T., male, aged forty-three years; manufacturer; American. Complains of headaches and sharp pain over right temporal, malar, superior dental region and lateral right nose, continuous for past ten days. Before that every week or two for a day during past few months. Cannot sleep or eat. Frequent colds, obstructed nasal breathing, excessive nasal discharge. Has been advised to have nose operated for relief of symptoms.

On inspection, nose presents engorgement of all mucous membrane, with great relaxation. Cystic right middle turbinate. No pus. Excess mucous nasopharynx. Pharynx, fauces, larynx and tracheal mucous membrane intensely injected, engorged and edematous from capillary transudation.

All retract to nearly normal position under stimulation. Referred to physiologist for physical examination, which shows: Man five feet six inches in height; weight, 150.5 pounds. Abdomen corpulent; cyanosis of extremities. Anxious expression to face—peripheral vessels show no arteriosclerosis. Capillaries chronically dilated about face, neck, upper abdomen and lower chest.

The larger nerve trunks are tender to pressure. The reflexes are moderately exaggerated, especially the knee jerk; the skin shows marked demography, no eruption. There is no lymphatic glandular enlargement; no edema of extremities. The tongue is coated throughout heavy white. The saliva is strongly acid to litmus. The lungs show no abnormality.

The heart, trifle hypertrophied, dullness just outside the upper line and in fifth space. There is a soft blowing systolic murmur at apex and transmitted to left as far as axillary line. The first sound at aortic area is impure, the second aortic is not accentuated.

Abdomen shows decided tympany over whole of colon and small intestine, stomach dilated to below umbilicus.

Tenderness over right temporal, malar and nasal regions.

Ophthalmoscopic examination shows overfilled veins and stasis in capillaries.

Urinalysis: Total thirty-two ounces, acid, 1022, no albumin, no sugar, no casts, marked indican.

Blood shows hemoglobin 90 per cent; leucocytes, 7000. Blood pressure: Systolic, 100; diastolic, 70 hgm.

This case was a typical example of gastrointestinal toxemia. Appropriate attention to the intake and output cleared up the whole situation unusually early. The trifacial irritation was especially aggravating, but was the symptom which cleared up earliest. The upper respiratory mucous membrane retracted and maintained normal position and function.

Case 2.—F. B., male, aged fifty-seven years; American; married; employed in general postoffice. Complains of hoarseness two months. Has been able to only whisper at times. Nasal breathing space decidedly lessened. Good deal of secretion from nose and throat. Family and personal history negative.

Examination: Nose: Considerable venous stasis and relaxation. Some clear mucus on floor posteriorly. Pharynx and fauces more relaxed than engorged. Larynx: Rima and arytenoids congested; false cord infiltration, considerably more in right than in left. Vocal cords greatly engorged, juicy and irregular in outline. On phonation, right cord moves past center posteriorly; absolutely no movement of left cord, which remains in cadaveric position.

Decided to have chest X-rayed and general condition investigated.

Physical examination shows patient anemic, pulsating brachial arteries. Small vessels show stasis, especially upon abdomen and lower chest. Hands are cyanotic and cool. No general lymph gland enlargement. Reflexes, knee and arm, are increased.

Skin is poorly nourished, but shows no edema or eruption. Demography is marked.

Lungs show good respiratory murmur over whole chest.

Heart slightly hypertrophied; no murmurs are heard at apex or base. Second aortic sound is greatly accentuated. The dullness across the base of great vessels is increased to the amount of three centimeters. There is a lack of muscular sound to the first sound at apex.

Abdomen tympanitic, especially stomach and small intestines. No masses palpable. Liver of normal size, but tender. Spleen negative. Stomach is distended with gas; extends to umbilicus.

Tongue is coated and larger than normal; shows indentation of teeth.

Peripheral nerve trunks tender to palpation.

X-ray shows enlargement of tracheobronchial group of lymphatic glands with calcareous degeneration of some of the bronchial group.

Urinalysis: Acid, forty ounces total in twenty-four hours, ten grains urea to ounce; no albumin, no sugar, no casts; indican increased.

Phenosulphonaphthalin test, first hour 32.5 per cent, second hour 27.5 per cent, third hour 10.0 per cent.

Weight 194 pounds formerly, now 184 pounds.

Blood examination: Hemoglobin, 85 per cent; leucocytes, 8400.

Blood pressure: February 23, 1914, systolic, 190; diastolic, 140. September 17, 1914, systolic, 152; diastolic, 90.

In the case of F. B., the main complaint was the hoarseness. No local treatment was advised. Attention to the cardiovascular and gastrointestinal changes cleared up the laryngeal lesion absolutely in about six months. At present the vocal cords are acting normally, the mucous membranes of upper respiratory tract in normal position, and the general condition of the patient is vastly improved.

Case 3.—Mr. H. V. T., aged thirty-three years; American; married; flour broker. Complains of being down in general condition for past six months—inability to breathe through nose and hacking cough. Has lost fifteen pounds in past nine months. Good deal of secretion from nose and throat. Eight years ago had congestion of left lung. Family and personal history otherwise negative.

On examination nose presents great venous stasis; mucous membrane blue, engorged and macerated; mucopus from left infundibulum. Retracts to nearly normal position under adrenalin. Slight anterior convexity of cartilaginous septum. Pharynx presents small nodules of lymphoid, no obstruction. Moderate drip posterior choanæ. Fauces moderately engorged. Larynx—false cords and rima greatly engorged and reddened. Vocal cords hyperemic and edges roughened; parallel on phonation. Posterior wall somewhat thickened, but not characteristic of tuberculosis.

Advised to have sputum examined and general condition investigated.

Physical examination shows man well nourished, venous stasis throughout body; likewise capillary. Peripheral blood vessels show no connective tissue change or muscular spasm. There is a general glandular enlargement of anterior and posterior cervical chain, axillary and inguinal.

The skin shows no eruption, but gives a good reaction in form of demography.

The tongue is heavily coated, especially the posterior one-third.

The chest shows incomplete consolidation of whole left upper lobe and moderate infiltration of the apex of left lower, especially posteriorly. The remainder of chest is negative.

Heart dullness not increased; sounds weak at apex and base; decided lack of muscular tone to first sound. No murmur heard.

Abdomen corpulent, tympanitic throughout.

Liver trifle enlarged and very tender to palpation. Spleen not felt.

Temperature, 99.4° F.; pulse, 120; respiration, 18.

Urinalysis: Acid, 1020; no albumin, no sugar, no casts; indican moderate.

Blood shows hemoglobin, 88 per cent; leucocytes, 10,000.

Blood pressure: Systolic, 118; diastolic, 90.

Von Pirquet reaction for tuberculosis positive. Sputum shows tubercle bacilli.

In this case the attention of the patient was centered upon the nasal obstruction and cough secondarily. As above findings show, case was of bacterial toxemia.

No local treatment was advised, but the general pulmonary lesion was at once attacked, with resulting healing. Four years later the patient was in fine condition, with none of the original venous stasis of the upper respiratory mucosa.

LXVII.

TWO MAXILLARY SINUSES IN ONE MAXILLA.

BY

O. H. Horrall, A. B., and H. F. Rawlings, M. D.,

Chicago.

This anomaly was found in the left maxilla of a white man of about fifty years of age. In this left maxilla we found two sinuses, an anterior and a posterior, separated by a complete osseous septum. The sinus in the right maxilla is normal, with a capacity of 2.4 centimeters.

The septum which separates the two sinuses is in the frontal plane above the middle of the third molar tooth. It is about 0.25 millimeter in thickness, thirty-one millimeters vertical measurement and sixteen millimeters mediolateral measurement. It is smooth and covered by mucous membrane on both sides. The more anterior of these two sinuses represents the normal sinus maxillaris (antrum of Highmore). It communicates by means of the infundibulum ethmoidale with the middle meatus. The frontal sinus, which is rather large, extending in the orbital bones two-thirds of the way over the orbital cavity, open normally into the anterior part of the infundibulum ethmoidale. Three anterior ethmoidal cells open by separate ostia into the upper part of the infundibulum. The anterior maxillary sinus has no pockets and no accessory ostium. The capacity of this sinus is 7 centimeters; greatest vertical extent, 34 millimeters; greatest mediolateral extent, 24 millimeters; greatest anteroposterior extent, 24 millimeters. The opening of the sinus is elliptical; the greater diameter, anteroposterior, 5 millimeters; the lesser, vertical, 3.5 millimeters.

The posterior sinus communicates by means of a single ostium with a small but distinct cell. This small cell, which is obviously a posterior ethmoidal cell, communicates with the inferior posterior part of the superior meatus by means of an ostium which is 12 millimeters anteroposteriorly by 3 millimeters vertically. The ostium between the posterior sinus and this cell is 3.2 millimeters anteroposteriorly by 7.4 millimeters vertically. The capacity of the posterior sinus is 4.35 centimeters; greatest vertical extent, 33.5 millimeters; greatest mediolateral extent, 16 millimeters; greatest anteroposterior extent, 22 millimeters. This posterior sinus has three distinct pockets, one in the superior anterior part, one in the superior posterior part, and a third in the inferior posterior part. Each of the two superior pockets is formed by a partial osseous septum projecting downward from the superior surface, while the inferior pocket is formed by a septum rising from the floor of the cavity. These incomplete semilunar septa project into the cavity not more than five millimeters.

The abnormal posterior sinus is clearly an aberrant posterior ethmoidal cell, which has invaded the body of the maxilla and there developed extensively.

All measurements were made with the mucous membrane, which lines the sinuses, intact.

The specimen is preserved in the anatomic museum of the University of Chicago.

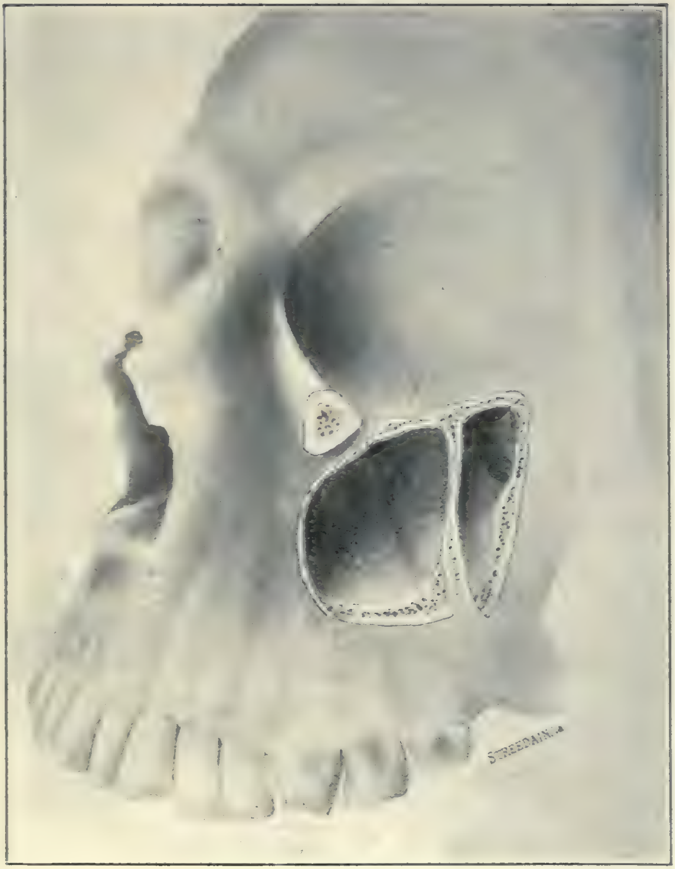

FIGURE 1.

Left maxilla, showing the two sinuses described in the text, and the partition separating them.

LXVIII.

THE ADVANTAGES OF THE DENKER OVER OTHER OPERATIONS ON THE MAXILLARY ANTRUM, WITH A REVIEW OF TWENTY-ONE CASES AFTER THIS METHOD.

By W. Likely Simpson, M. D.,

Memphis.

There is, of course, not such a thing as any one operation being perfect for operating on all chronic suppurating antrums, but the Denker operation has, it seems to me, more to recommend it than any other operative procedure which has been suggested up to this time. I shall not compare this operation with those procedures in which a temporary opening is made in the inferior meatus, middle meatus or both for the relief of acute suppuration in antrums without polypi, necrosis of bone, etc.—in other words, in antrums with only a moderate pathologic process. It is the opinion of some that in antrums with only moderate pathologic changes, washing the cavity through the inferior meatus will cure the great majority of these cases without any operation.

If an operation is to be recommended for relief of chronic antrum suppuration, it should be a procedure with the following advantages:

1. It should give as a final result a normal functionating nose without a tendency of antrum suppuration to relapse.

2. It should allow the operator to be thorough in the removal of all polypi, necrotic bone, etc. All parts of the antrum should be opened to direct inspection.

3. It should leave the nasal cavity as near as possible in a normal condition. The turbinates should not be sacrificed, and too large an opening should not be left between the antrum and the nose cavity, so that an atrophic condition would be set up.

4. It should have a healing period reasonably short and without any considerable pain or discomfort.

5. It should have few or no complications, as fistulas into the mouth, lacrimal obstruction, etc.

The advantages of the Denker operation over other procedures recommended for relief of extensive pathologic changes in the antrum can probably be best shown by comparison with each individual operation.

The alveolar or Cowper operation has, it seems to me, nothing to recommend it. If an antrum suppuration is caused by a bad tooth and is acute or subacute, all that is needed is treatment of the tooth by a dentist and washing of the antrum through the inferior meatus by the rhinologist. If the antrum is a chronic one, then the removal or treatment of the offending tooth with a Denker operation on the antrum would be indicated. If a large opening into the antrum is made through the tooth socket, the after-treatment is long, probably a lifetime; the antrum probably becomes infected from the mouth cavity, and often it is necessary to insert a tube from the antrum draining into the mouth. To have a constant drainage of mucopurlent discharge into the mouth is very annoying to the patient, while a Denker operation, after the proper care of teeth, would bring about a cure in a very short time. In the Cowper operation the contents of the antrum cannot be inspected thoroughly, polypi and diseased bone cannot be removed thoroughly, as in the Denker operation.

The Küster operation, which consist in making a large opening into the antrum through the canine fossa, and clearing out granulations, polypi, etc., without making an opening into the nose and carrying out the after-treatments through the opening in the mouth, is not to be recommended. The only thing which the operation has to recommend it is that it allows inspection of the greater part of the antrum cavity. The Denker gives much better inspection, closes the opening into the mouth at once, gives perfect drainage into the nose, and the after-treatment is much shorter.

The points of advantage of the Denker over the Luc-Caldwell are as follows:

1. The opening into the antrum from the nose is farther forward in the Denker, making the after-treatment easier; especially is this so if the patient is to wash the antrum with a tube.

2. The anterior angle can only be reached through an operation which removes the bone from pyriform aperture posteriorly, opening the anterior angle. This is not done in the Luc-Caldwell operation. The inferior turbinate, if normal, is left intact in the Denker, while in the Luc-Caldwell the anterior part is usually removed; therefore, in this respect the Denker would be much preferred. The mucous membrane in the nasoantral wall in the Denker, which is laid on the floor of the antrum, encouarges a final good result, and therefore shortens the time of healing very much. This flap also prevents the piling up of granulations at the base of the bony nasoantral wall, which is very liable to occur in the Luc-Caldwell.

The Denker operation has all the advantages of the Luc-Caldwell, with the other advantages first mentioned.

The only disadvantage which I have seen from the Denker operation is that the nerve filaments to the central incisors and canine teeth are cut, causing a numbness in these teeth; but it has been my experience to see these symptoms gradually become less marked and finally not noticed by the patient.

The Canfield or Canfield-Ballenger operation has some of the advantages of the Denker, but certainly an intranasal operation on an antrum filled with polypi, etc., is not as satisfactory as the Denker, where direct inspection through a good-sized opening can be obtained. The Skillern operation is very similar to the Canfield, but is less radical, and really cannot be recommended for chronic suppuration.

The intranasal opening of the antrum as suggested by Myles, the operation of Krause, removal of anterior part of the middle turbinate and making quite a large opening into the antrum, also making a large opening into the antrum with saws after the removal of the anterior part of the inferior turbinate, as suggested by Vail; the opening into the antrum with chisels, as suggested by Stein, Corwin, etc.; trephining of nasoantral wall by Bishop, and rasping of same by Wells, and cutting away of wall with Ostrom's forward cutting forceps, the breaking down of a larger part of nasoantral wall, with retention of inferior turbinate, as suggested by Sluder; the saving of inferior turbinate, as suggested by Hirsch, with a large opening inferior to the turbinate; the operation of Rethi, in which a large opening into the antrum through both inferior and middle meatus, and Roe's opening of nasoantral wall with forceps, and the operation of Freer, etc., are all good for mild cases, but if the antrum is filled with polypi, the bony walls are necrotic, or cysts are contained in the antrum, then only a more radical operation is indicated, and I believe the Denker is to be preferred.

It has been said that there is too much shock to the external operation, and that it is necessary to operate with a general anesthetic, but I do not find that either of the statements hold true. The Denker can be done with very little discomfort with cocain and novocain and without shock. As a rule, I do not find it necessary to use a general anesthetic. There is no more necessity for a general anesthetic for the Denker operation than there is in removing a crooked bony septum of the nose.

It seems to me that the advantages of the Denker operation should again be mentioned:

1. It gives a good view of the interior of the antrum—better than can be obtained through an intranasal operation.
2. It gives a large opening at the anterior angle of the antrum, which is not obtained by any other external operation.
3. It gives a large mucous membrane flap, which materially shortens the after-treatment and prevents granulations from closing the opening from below.
4. It leaves a normal functionating turbinate and nasal cavity, usually healing from three to four weeks.

REVIEW OF CASES.

In reviewing the twenty-one operations, I shall discuss them briefly from the following viewpoints:

1. Anesthesia used.
2. Whether the antrum suppuration was complicated by other sinus troubles, diseased teeth, cysts, etc.
3. If other sinus troubles were present, was an operation done on this sinus at the same sitting, or previously, or afterwards?
4. Whether previously an operation had been done on the antrum.
5. Whether the wound in the mouth was entirely closed with sutures at the time of the operation.

6. If complications, what they were.
7. The final result.

1. As to Anesthesia.—In twenty-one operations, cocain and novocain was used in twenty cases, and ether in one. One per cent novocain was injected over the anterior surface of the antrum under the periosteum, after applying a small amount of cocain to the mucous membrane. I usually apply cocain with an applicator to the nasoantral wall and inferior turbinate. A solution of novocain and adrenalin is applied to the antrum cavity during the operation. If one is operating on a child or nervous woman, it would probably be best to use a general anesthetic, ether preferred. I used morphin, one-quarter grain, about one hour previous to the operation in my former cases, but of late have discarded it and do not see any difference in the discomfort.

2. Complications of the Antrum Disease by Suppuration in Other Sinuses, Cysts, etc.—Only two of these cases were complicated by frontal sinus suppuration, and ten of the operations were complicated by ethmoid suppuration. One case had a sphenoid suppuration. None of the cases was complicated by cysts, bad teeth or bony septum in the antrum.

3. If Other Sinuses Were Affected, Was an Operation Done on the Other Sinuses at the Same Sitting as the Denker Operation, or before or after?—In only one case was the middle turbinate removed and ethmoid curetted at the same sitting of the Denker operation. In ten cases the middle turbinate was removed and ethmoids were curetted previously to the Denker, and in one case the sphenoid was opened previously to the Denker. In two cases an intranasal frontal sinus opening was made after the Denker.

4. As to Whether a Previous Operation had been Done on the Antrum.—In no case was an operation done on the antrum previously.

5. As to Closure of Mouth Wound.—In ten cases the antrum was packed rather tightly; also on top of the nasoantral flap, and the end of the packing left hanging into the mouth through the mouth wound, and in two to three days the packing was withdrawn through the mouth and then two or three sutures inserted. This procedure is supposed to disturb the flap of the antrum less than if the packing is removed through the nose.

In the last eleven cases very little packing has been used and the mouth wound has been entirely closed, getting primary union, doing away with any danger of fistula in the mouth. Very little packing is necessary.

6. As to Complications.—In one case in an old gentleman, with a double Denker operation, erysipelas set in on the fourth day. The healing went along nicely, except that the lip was so swollen that sutures could not be placed in the mouth wound after the packing was removed and, therefore, fistulas in the mouth on both sides was the result. These fistulas, after some difficulty, were closed.

In one case a man with very filthy mouth and filthy habits, a small abscess of the soft part anterior to antrum was the result. After a few days this pus cavity was evacuated and he made a good recovery.

7. The Final Result.—I have followed seventeen of these cases and in each one there has been a good result. There has not been a relapse in one case. In one case, in which part of the inferior turbinate was removed, there is slight crusting, but the patient also has some ethmoid trouble which would account for that. In the cases without other complicating sinuses, the nose is perfectly normal and I feel that I am justified in being enthusiastic about the Denker operation.

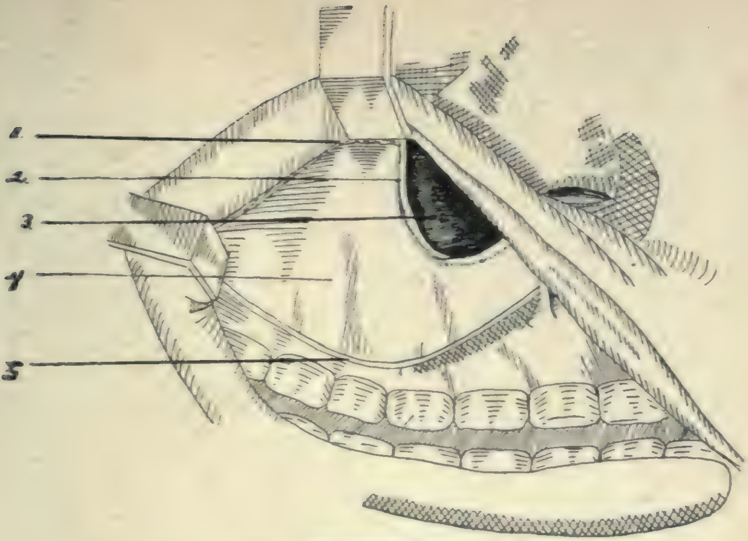

FIGURE 1.

First step in the Denker operation. The soft parts incised and retracted.

1—Lip and soft parts from the anterior wall of antrum, etc., retracted upward.
2—Apertura pyraformis.
3—Nasal mucous membrane.
4—Bony anterior antral wall.
5—The lower border of the incision.

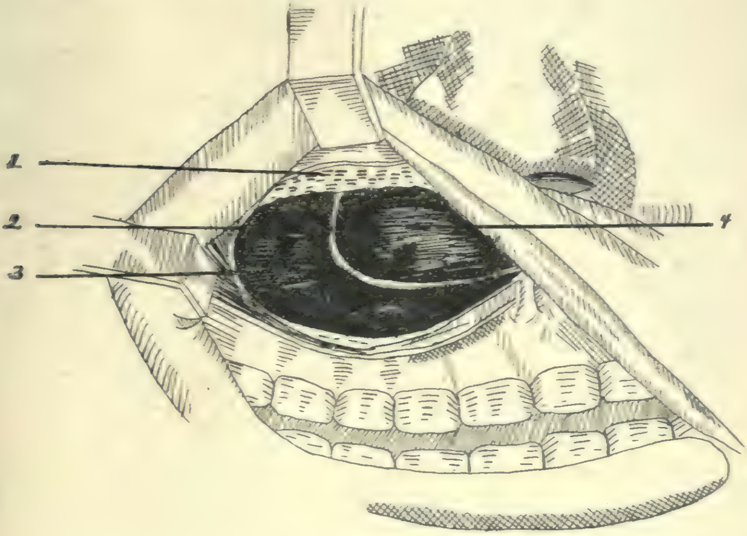

FIGURE 2.

Second step in the Denker operation.

1—The superior border of the removed bony anterior wall.
2—The border of the partially removed bony nasoantral wall.
3—The maxillary antrum.
4—The mucous membrane of the nasoantral wall.

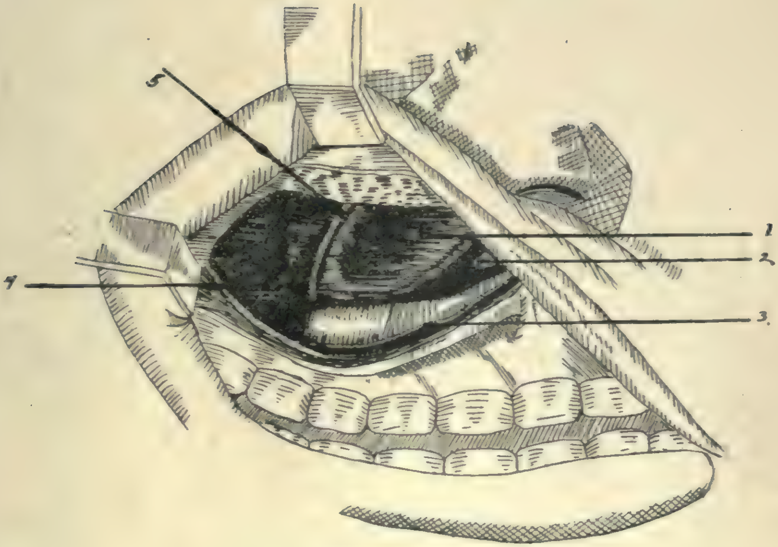

FIGURE 3.

Third step in the Denker operation.

1—The inferior turbinate body.
2—Space between the inferior turbinate and the flap opening into the nasal cavity.
3—The flap of mucous membrane from the nasoantral wall lying on the floor of the antrum.
4—The maxillary antrum.
5—The posterior border of the partially removed bony nasoantral wall.

SOCIETY PROCEEDINGS.

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLOGY.

Meeting of April 14, 1916.

Scalp Infection Simulating Mastoiditis.

Dr. J. Morisette Smith: The patient, J. M., aged thirteen years, had otitis media purulenta acuta three weeks ago. A myringotomy was followed by a discharge which ceased in three or four days.

Three days ago a swelling was noted behind the right ear.

Examination showed a swelling over the mastoid tip resembling a Bezold perforation, with tenderness over the tip and behind in the region of the emissary vein. Dry drum membrane, bulging in upper posterior quadrant. No glands palpable; no evidence of furunculosis; nose and throat negative; no temperature.

The treatment consisted of rest in bed, cathartic, incision of drum membrane, boric irrigations.

X-ray plates, taken by Dr. George S. Dixon, showed diffuse cloudiness on right side.

The aural discharge stopped in two or three days, the swelling behind the ear disappeared, with some glands palpable. It was evidently a postauricular adenitis with an obscure focus of infection—probably the scalp.

DISCUSSION.

Dr. Hays said that when the girl's ear drum was opened up, before she was seen by Dr. Smith, there was only a slight discharge, and it cleared up nicely. On his return, there was apparently a mastoiditis, and the tip was tender; this condition was corroborated by the X-ray plate. The chief point of interest in the case was the conclusion that one must not put too much confidence in the interpretation of the X-ray picture in such cases without clinical confirmation. In this instance the X-ray findings were clear, and yet the cloudiness was due to a swelling over the mastoid. The best evidence that it was not a mastoid condition was that it cleared up in a short time after the patient's scalp was attended to. The glands subsided, and she has had no further trouble.

A Case of Acute Mastoiditis With Glycosuria. Mastoidectomy Under Novacain, Followed by Hyperpyrexia, the Latter Falling Synchronously With the Disappearance of the Glycosuria.

DR. LESTER MEAD HUBBY: S. M., male, aged thirty-six years. Had variola in infancy; otherwise no serious illness. No history of previous ear disease.

Nine days prior to admission had an attack of influenza. Six days later otalgia began. Within twelve hours otorrhea set in. Two and a half days later patient came to Dr. Phillips' clinic at the Manhattan Eye, Ear and Throat Hospital.

There was a profuse otorrhea, some narrowing of the external auditory canal near the annulus, marked tenderness over the lower half of the mastoid process, considerable pain near the mastoid, marked general prostration.

He was admitted for operation on the following day, with the following status: Temperature, 99.4° F.; pulse, 100; respiration, 26. Urine contained sugar, acetone, diacetic acid, albumin and casts. General condition poor.

Operation under local anesthesia was, therefore, deemed advisable. A hypodermic of morphin, one-fourth grain, was given half an hour before the operation.

Novocain, one-half per cent, was used intra- and hypodermatically, also hypoperiosteally, over the mastoid process and the posterior meatal wall of the external auditory canal. Novocain ten per cent was swabbed occasionally in the wound whenever there was evidence of slight pain. Practically no pain was produced except at the end of the operation, on biting off with a rongeur a rough edge of bone near the surface on the posterior edge, when a severe twinge occurred.

A very large mastoid was found filled with pus, and the dividing walls of the cells were partly necrosed. On culture the offending organism, taken from the mastoid wound at the time of the operation, was found to be the streptococcus hemolyticus.

The patient seemed to be none the worse after the operation, yet in three hours his temperature rose from 100° to 104° F., and remained around that point for about seventy-two hours.

During that period of seventy-two hours the following conditions prevailed: The urine contained sugar 3.3 to 3.5 per cent; acetone and diacetic acid were present; albumin .2 grains per liter; hylin and fine granular casts; some pus cells. Severe cephalalgia, especially in the vertex; at times a severely burning sensation there. Morphin occasionally and ice bag continuously were used for its relief. Very little nourishment taken.

The wound was rather loosely packed with iodoform gauze, so that there was considerable oozing of blood during the first forty-eight hours.

There was considerable cellulitis and tenderness below the mastoid at the end of this period.

The dressing was removed, the wound packed with sterile gauze, and a continuously wet dressing applied at noon April 27th—i. e., a day and a half after the operation.

No symptoms referable to the thoracic or abdominal organs were elicited by repeated examinations, except the urine findings.

There was no cough, no thoracic pain, no alteration of the pulse or respiration ratio.

Blood culture, taken on the 27th, was negative after thirty-six and ninety-six hours. (Dwyer.)

Leucocyte and differential count on the 27th was as follows: Leucocytes, 16,000; large mononuclear lymphocytes, three per cent; small mononuclear lymphocytes, 10 per cent; polynuclear neutrophiles, 87 per cent.

On the evening of the 28th, seventy-two hours after the operation, the temperature started to fall by lysis; the sugar, acetone and diacetic acid disappeared from the urine on the following day, and the patient gradually returned to a normal condition.

He left the hospital on the fourteenth day with a normal temperature. He returned every other day for change of dressings.

The mastoid wound healed very slowly. Near the tip the wound closed and opened many times for about six months.

Since leaving the hospital the urine has been examined periodically, and only twice at long intervals has there been a very slight trace of sugar.

The puzzling feature of this case is the postoperative temperature. Aside from the temperature curve there were no symptoms of pneumonia. Unfortunately, the X-ray was not being used at that time to demonstrate hidden pneumonias.

Absorption of fibrin and other ferments from a wound sometimes causes a high temperature, but the temperature in this instance continued after the disappearance of the hemorrhagic discharge.

URINALYSES.

	April 27th	28th	29th	30th	May 1st	2nd
Spec. grav.	1028	1032	1020	1022	1024	1010
Reaction	Acid	Acid	Acid	Acid	Acid	Acid
Albumin per litre......	.2 gms.	.2 gms.	.2 gms.	Trace	Trace	.1 gm.
Sugar..............	3.5%	3.3%	Neg.	Neg.	Neg.	Neg.
Bile................	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.
Blood..............	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.
Urea (gms. per liter)	16	18	16	14	18	....
Indican.............	Trace	....	Trace	Sl. trace	Trace	Sl. trace
Casts...............	Hyaline Fine gr.	....	Trace	Sl. trace	Trace	Sl. trace
Blood cells..........	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.
Pus.................	Some	Some	Some	Some	Mod'rate	Some
Crystals............	Uric ac. Calc. ox.	Some	Some	Some	Calc. ox.	Calc. ox. Uric ac.
Bacteria............	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.
Acetone.............	Present	Neg.	Neg.	Neg.	Neg.	Neg.
Diacetic acid.........	Present	Neg.	Neg.	Neg.	Neg.	Neg.
Amount 24 hrs.............		750 cc.	......	750 cc.	450 cc.	620 cc.

The cellulitis in the neck would hardly produce such an even temperature curve.

The disappearance of the sugar at the moment of the lysis of the temperature curve was also interesting; whether this was due to the starvation of the first day or two, followed by a diabetic regime, or directly to the disappearance of the cause of the sugar excretion, it is difficult to say.

The sugar excretion in diabetics is liable to be increased by anxiety or shock, and it is to be noted that this patient has a very neurotic diathesis.

This may, therefore, explain the glycosuria at the time of this illness.

The examinations on May 3rd, 4th, 7th and 9th were about the same as those of May 2nd, except that the amount of urine excreted steadily increased.

There was no discussion of the case, but Dr. Hubby expressed his regret that he had not taken an X-ray of the chest in order to absolutely rule out pneumonia. Excepting the temperature curve, there was no symptom of pneumonia.

Paper: Extensive Keloid of Mastoid Wound Cicatrix.*

BY FRANK L. RYERSON, M. D.

DISCUSSION.

DR. GRAEF said that it was an exceedingly interesting case. He had seen something like it on the lobule of the ear, from wearing ear rings. Some years ago, in London, he saw some interesting keloids in the clinic of Jonathan Hutchinson, who said they often came from flogging in the old days, and showed two cases. It would seem that these patients have a peculiar excitability of the skin, as there is nothing malignant about the growths. Perhaps Dr. Ryerson could make an experimental cut or two somewhere on the body and see if keloid scars developed, or if, perhaps, the long continued suppuration was a factor in this particular location.

DR. HUBBY said that three years ago he had presented a case of keloid of the auricle which had been removed twice and recurred. He removed it on the second occasion, and then, as it began to recur, turned the patient over to Dr. Law, who treated it with the X-ray for six months and stopped the growth. Dr Hubby said he did not remember how often the treatment was administered, nor the dosage, but the condition had not recurred. He had seen the patient about two months ago.

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*See page 947.

Dr. Leshure said that he had had a case something like this one, occurring in a mastoid wound; it was like this one in that it followed a long continued suppuration. He asked if Dr. Ryerson had tried the high frequency current; that is sometimes beneficial.

Dr. Paule Bechet said that the cause of keloid was not known, and that while spontaneous keloid arising from an absolutely unbroken surface was mentioned in textbooks, it was his opinion that it invariably followed some break of the skin surface, no matter how slight. He had frequently seen keloid follow the attempts of patients to express comedones on the chest. The lesion in Dr. Ryerson's case was typical keloid, and bore no clinical resemblance whatever to sarcoma. It might be of interest to mention that the main point of differentiation between keloid and hypertrophic scar was that the former always extended beyond the limits of the original trauma. The treatment of this condition was disappointing; of all the therapeutic agents, the X-ray gave the best results. It should, however, be given in measured doses and pushed to the point of erythema. The massive dose method was particularly applicable. The old-fashioned fractional dose method with unmeasured rays was practically useless. With X-ray treatment the tumor flattened, lost its hardness, and in favorable cases disappeared, leaving a soft, smooth scar, but unfortunately was frequently complicated with telangiectasia, a condition which frequently follows the use of the X-rays. Internal treatment with arsenic, etc., was useless. Fibrolysin had been tried, and had not given any remarkable results. A ten per cent solution of thiosinamin, in equal parts of glycerin and water, or in alcohol, of which ten to twenty minims are injected into the growth, has, according to the reports, given excellent results. Excision is not advisable, as recurrence almost invariably takes place. Where the growth has been very small, electrolysis has been very successful.

Dr. Ryerson, closing the discussion, said that as yet no histologic examination had been made, but he would have this done later on. He wished the section to see the patient before anything was attempted for his reliet. The man had a number of scars on his fingers, but none on his body. The high frequency current has not been applied.

Dr. Ryerson said Dr. Bechet seemed to think he had the idea that keloids could occur spontaneously, but he had not meant to convey that impression. It may have its origin from a scratch which has been forgotten.

Report of a Case of Mastoiditis Complicated by Suppurative Arthritis of the Shoulder. No Sinus Involvement.

DR. JAMES P. ERSKINE (by invitation) said that the main points in the history were the development of a mastoiditis following influenza. The patient had been confined to bed for a week, and then after having had earache for a day and a half he examined the ear and found a bulging drum with a hemorrhagic bullæ on the membrane. The drum was incised and drained. He saw the patient at the end of a week, and he seemed to be better for five days, and then for two days again suffered with earache. His physician said his characteristic pulse was 64. Five days after the incision he had more pain, enough to keep him awake at night, and he was not a man who was given to make undue complaint. A culture showed streptococcus brevis. There was then some tenderness over the mastoid, so it was recommended that the mastoid be opened. The temperature at that time was 100° F.; it did not go above 101° F. The family hesitated for three days about having the operation performed; but finally, ten days after the earache, the mastoid was opened. On that afternoon there was a sudden rise of temperature to 103° F.; previous to that it had not been above 101° F. There was nothing unusual about the mastoid operation. The mastoid was large. The man was large, was over six feet tall, and weighed one hundred and forty-seven pounds. He was below par; an intimate friend said that he had been drinking a good deal and eating very little. There was free pus. The cells were extensive. The dura was exposed for a small area—about one by one-half centimeter.

On coming out of the anesthetic the man complained of pain in the shoulder of the opposite side. Dr. Erskine said that he had followed the man down to the ward from the operating room, and was confident that there was no roughness in handling him; but he declared that his shoulder felt as if it were dislocated. There was tenderness over the front of the shoulder, and the movements were painful. The next day it showed redness, and three days later, when the joint was opened, the arm was very much swollen. The chief swelling was in front. The temperature was 102° to 103° F.; the man had profuse perspiration, but no chills, and no drop in the temperature. He had a practically uniform temperature, and was very, very ill. He was very uncomfortable, and was obliged to lie on his back, the slightest movement of the arm causing exquisite pain.

Dr. Erskine said that his first thought was that the condition of the shoulder was a secondary metastasis from a sinus thrombosis. The blood culture was negative. The headache and other symptoms disappeared after the operation. He then thought of meningitis, but the spinal fluid was clear and negative. The leucocyte count was 23,900, and 83 per cent polynuclear count. It was then thought that there was not a new focus—the temperature being the same as before operation—and that the joint was the chief condition. A general surgeon opened the joint and found three ounces of serous pus. The culture from the mastoid showed streptococcus, and the culture from the pus in the shoulder also showed streptococcus, and was said to be similar to that from the mastoid; also the pus taken from the ear before the mastoid showed streptococcus.

Immediately after the incision of the shoulder joint the temperature went down. Two days later an inflammation like erysipelas developed, but the temperature was dropping. Dr. Erskine said he would be glad to know if the members thought this was an erysipelas; he did not investigate it at the time, for he feared the patient might be put out of the hospital, but the surgeon agreed that it was erysipeloid in character.

The patient's subsequent course was uneventful, and he recovered. The drum discharged for six weeks.

Dr. Erskine said that this case kept him on the anxious seat for three days. It was evident that there was a suppurative arthritis of the shoulder. The question was whether the patient should be given the benefit of early operative treatment for a possible sinus thrombosis. As he was so ill, it seemed better to drain the shoulder first. As the temperature dropped there could not have been any sinus thrombosis.

Dr. Graef did not think the ear had anything to do with the shoulder condition. There were two independent infections. He had only that day seen a boy of fifteen years, who had been operated upon on December 15th for a submucous resection, and who three days later developed a septic arthritis of the knee joint. The joint was opened and treated, and the boy will have a stiff joint the rest of his life. That submucous operation was done in a well-known hospital, and no doubt carefully. If sepsis could thus invade the knee joint, why not the shoulder, as in Dr. Erskine's case? Both the mastoid invasion and that of the shoulder joint might have been traced to an infective focus in the throat, teeth, or nose, both occurring at the same time.

Dr. Opdyke told of the case of a boy with a mastoid in the right ear and arthritis in the elbow, knee and ankle joint; a year later he had a mastoid on the other side, and had the same involvement of the other ankle, knee and opposite elbow. It was a simple mastoid, and there were no signs of lateral thrombosis at either operation, but he had a long convalescence. The ankle, knee and elbow were not opened in either instance, although they were involved for a long time.

Paper: A Modified Radical Operation for Chronic Suppurative Otitis. Lantern Slides and Demonstration of Patients.*

By Hugh B. Blackwell, M. D.

DISCUSSION.

Dr. Chambers said that he did not clearly understand what the operation was and wherein it differed from any other mastoid operation, except in omitting to remove the incus and malleus, diseased or healthy.

Dr. Yates said that on Randall's Island they had been trying to solve pretty nearly the same problem in children under fifteen years of age. This problem was to clear up the ears with as little after-treatment as possible and still preserve useful hearing. They had tried every operative procedure that had been suggested and some that had not. The difficulty with modified radical operations had always been that after bony dissection had been carried down to the tympanum, the ossicles were found to be embedded in a mass of granulations or swollen mucous membrane, so that it was impossible to determine the condition of the conductive auditory apparatus. Therefore, they were often impelled to finish an operation, planned on conservative lines, as a complete radical, rather than leave an undetermined pathologic condition behind.

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*See page 908.

So far as functional results were concerned, he was inclined to think that the radical, with skin grafting, gave as good results as anything that had been done. The Heath operation had been tried out, with flap cutting and without. Simple mastoid exenteration, combined with ossiculectomy or the Yankauer procedure had also been employed in appropriate cases with very satisfactory results. But there were many cases which must be classed simply as chronic suppurative mastoiditis. These drain through the middle ear without doing much damage to the latter, and if the focus in the mastoid was removed by a simple mastoid operation, taking care to remove all diseased bone in the region of the aditus, prompt healing with excellent function would result. Flushing out the tympanic cavity at the end of the operation seems to hasten recovery in such cases.

Dr. Yates also asked what proportion of perforations of the drum membrane healed after Dr. Blackwell's operation.

Dr. Guentzer said that Dr. Blackwell had not stated how he discriminates; it would seem that he selects those cases with a drum membrane, with a perforation. Bearing that in mind, it might not be fair to compare the results with the radical operation, as it is usually understood; and the hearing might be expected to be better than after a radical operation.

He would like to know whether Dr. Blackwell performed this operation on the ordinary cases where the ossicles were destroyed by necrosis.

Dr. Braun asked Dr. Blackwell whether he performed his operation in cases with cholesteatoma, and in how large a proportion of cases, he succeeded in getting a dry ear.

Dr. Hays said that most of the men had gotten away from the old idea that it is necessary to do a complete radical operation in every instance, and most men employ some more conservative method in many cases. In many instances he is himself satisfied with cleaning out the attic and antrum, and disturbs the middle ear as little as possible, and makes no flap at all—simply making a communication between the antrum and mastoid and middle ear.

Two months ago he did an operation on a girl who had had a discharging ear for six years. The girl's ear healed completely after taking down the posterior wall, and the hearing had considerably improved. In this case the dura and antrum, and the sinus and the middle ear were all within an area of a cubic centimeter, and it was very difficult to work upon, so it was thought best to do as little as possible, and allow the ear to heal up. In a number of such cases one can be quite conservative and yet get good results with chronic discharging ears.

Dr. McCullagh, referring to the Heath operation, said that if his memory served him, he had read a final report made by two Boston men on one case operated upon by Mr. Heath by his method and the end results were practically all unfavorable. In many of them it was necessary to do a secondary radical operation. As he understood Dr. Blackwell's method, it was very nearly akin to the Heath method, and the main objection to it was that it would not be applicable to many cases in which the radical operation is called for, in that it does not give an exposure of the middle ear.

He further said that in the cases in which he felt a radical operation was necessary, it was seldom that the incus was present. In a great percentage of cases needing operation, both the incus and the malleus are necrotic if present, and there is necrosis in the middle ear in regions which cannot be exposed by the precedure described. It seemed that most of Dr. Blackwell's cases were recent cases. It is well known that there are types of chronic discharging ear in which bone changes do not take place; these cases seem to be favorable ones for the Yankauer operation, which is even more simple than the one which Dr. Blackwell advocates. It would be interesting to know how Dr. Blackwell determines in what cases it is safe to leave the ossicles in place, and how he is sure that he has removed all necrosed bone.

Dr. Blackwell, closing the discussion, said that only one of the cases operated upon had cholesteatoma. In that instance the patient was a boy who had had a discharging sinus for two years, and there was considerable cheesy material in the ear. His ear has not been dry for three years.

So far as acute symptoms are concerned in the cases that had running ear for six months, there were no acute symptoms present, but the patients had headaches and dizziness. The big stout woman had had headache every day for six months, and was very dizzy; she was seen by a number of men in the clinic, and a radical operation was advised.

As far as the drum was concerned, he had found that where the drum was practically gone there was no use in attempting to save any remnant. He had performed this operation on a young girl with a double condition, and she can hear the acumeter at twenty feet in the ear that has scarcely any drum at all; so it would appear that even in such cases there is apparently some advantage in leaving the remnant of the drum in position and not cleaning out the middle ear.

The trouble he has had in the cases in the after-treatment was that the granulations have a tendency to reform and accumulate in the upper attic, where a thick plug would develop. In his opinion the best way to treat such cases was to pack the attic very tightly until the secondary drum membrane forms.

There was no fixed rule in selecting or treating these cases. The only attempt was to select such patients in the clinic as showed a certain amount of intelligence and responded to the qualitative tests for hearing.

DR. L. M. HUBBY presented a specimen of

Sequestrum of the Labyrinth.*

DISCUSSION.

DR. GUTTMAN asked how soon the compensation took place.

DR. HUBBY, closing the discussion, said that the patient was seen only a few times except when in the hospital. As he had stated, the process was a slow one, and the compensation proceeded step by step with the slow destruction of the parts of the vestibular nerve.

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*See page 980.

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLOGY.

Meeting of May 12, 1916.

Paper: The Social and Economic Aspects of Deafness.*

By A. J. Amateau.

BY INVITATION.

Paper: The Psychology of Deafness from the Standpoint of the Patient.†

By George W. Johnston, M. D.

The Partially Deaf Child.

Prof. John D. Wright said that he was always glad to have an opportunity of talking to persons who are interested in the deaf, for any reason, but was especially pleased to speak before the Otological Section of the New York Academy of Medicine, for these gentlemen were able to give the information to the parents of deaf children that they need at the time they need it. Every deaf child is taken to some physician, and the parents are guided by what he says in regard to the child. If the physician is intelligent and well informed concerning the deaf, he is able to deal with the child, not only from the specialist's standpoint, but when he has done everything that medicine or surgery permit, he can add still further to his service by telling the parents how to aid by education the imperfect organ with which the child must go through life.

The gentlemen who made out the program had shown unusual intelligence in dividing the subject into its three basal divisions as referring to the education of (1) those who are totally, congenitally deaf, or deaf from early infancy—the phase of the subject treated by Dr. Taylor; (2) those who are not totally deaf, but have a residual amount of hearing from infancy; and (3) those who have had the misfortune to lose their hearing after speech has been acquired. These are the various educational phases that are encountered in the schools.

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*See page 930. †See page 942.

Prof. Wright said that the phase which had been assigned to him could not possibly be covered in the brief time allotted, so he must content himself with trying to make two or three essential points clear. As most persons are more interested in what is seen than in what is said, he had brought three pupils from his school to illustrate some of the points which were most important.

First of all, it should be remembered, by the doctors especially, that a very slight degree of impairment of hearing is a far more serious matter to the child than to the adult. A degree of deafness which would not be a serious matter to an adult would be a grave handicap to a child, for it interferes with the acquirement of speech and language. It is surprising how very slight an impairment of hearing will interfere with the acquirement of speech. Even if the child's hearing is only very slightly impaired, it should receive special educational attention very early.

The next point of importance is to know how even a very slight amount of hearing can yet be made of service to a child. A child may have a very considerable power of hearing and, nevertheless, grow up without acquiring speech or language; and, on the other hand, with proper teaching, though only possessing a slight perception of sound, he can be given an approximately normal training. This reminded him of what the tramp who asked for work said when the lady replied that she had none for him: "Oh, ma'am! If you only knew how very little it would take to occupy me!"

The residual hearing, to be of any service, must lie within the range of the speaking voice. Undoubtedly it is worth while to spend time in training the residual hearing that lies outside the range of the speaking voices of men and women, but for education in language and speech, the only thing that is valuable to us is where the islands of hearing are within that range, and when this is the case a very little perception is valuable. If physicians and those interested in deaf children could only realize how very little hearing is required to fit a child to lead a normal life, many more of them would have this training.

Unfortunately, one of the children Prof. Wright had wished to present at the meeting was ill and could not come. She represented one of the deafest of the cases that can be dealt with educationally. She is a girl of thirteen years, and was eleven when she came to Prof. Wright's school nearly two years ago. She had been for three years in a large institution for the deaf and dumb, starting when she was eight years old. While those in charge of the institution probably recognized that she had some perception of sound, they could not give her the individual attention necessary, so nothing was done for her hearing. Her father realized that she had some residual hearing, though it was not enough to affect the modulation of her voice, and though she has now acquired a considerable ability to comprehend language through the ear, she still has the modulation of a totally deaf child. When she first came under Prof. Wright's training she could not distinguish any vowel sounds, could not distinguish any words spoken into her ears, could not, indeed, understand what words meant. For two years, patiently and continuously, she has received training—the education of the brain through the ear—and today the teacher is able to read the greater part of her history lesson to her. This training was begun very slowly, first with the vowels and then with words, and today she can understand a great deal and it has had a great effect upon her language. She has been examined as carefully as any physician can examine a deaf child, and has been pronounced totally deaf in the left ear, with only a very slight amount of hearing in the other, which, he states, is through bone conduction. However that may be, we can communicate with her almost as easily through one ear as the other.

The first example shown was a little girl who came to Prof. Wright last January, only four months ago, at the suggestion of Dr. Yankauer, who, unfortunately, was unable to attend the meeting. She is seven years old, and has a considerable perception of sound. After some months of treatment, Dr. Yankauer made up his mind that he could do nothing more for her surgically or medically, but that this perception of sound was very valuable to her if she could be trained. She had, however, reached seven years of age without having acquired any vocabulary. She knew a very few words—could say baby, papa, mamma, and auntie, but otherwise had no language, no speech. That was four months ago. Work has been done with her both through her eyes and her ears. She has been taught to associate a good many ideas with the imperfect sounds which she hears. She has to be taught a hearing vocabulary, just as she is taught a seeing vocabulary.

Ruth: (Demonstration. The teacher spoke directly into the child's ear and she responded, showing that she understood the sounds, although occasionally she was allowed the assistance of her eyes.)

The start is made with three sounds, a whistle, a bell and clapping of the hands, with the child's back turned. Sometimes it takes several weeks for them to distinguish between these three sounds. Then three vowels widely separated from each other in sound, and then these vowels in connection with words, putting the objects for which the words stand on the table and teaching the child to pick up the object named. The teacher first showed a few of the many words and sentences that the little girl has learned to understand through her ears, as, for example, "Shut your eyes," "Laugh," "Cry," "Brush your hair," etc. Then a few minutes were given up to showing rapidly the process by which the results had been obtained in the four months. First, the teacher spoke the vowels a, oo, ee, and the child repeated them. Then a toy car, and sheep, and a spoon were placed on the table, these words containing the same three vowels that were spoken, and the child pointed to the object named, then spoke the words herself. To teach the pronunciation of the words in the first place the help of the eyes were required. Then a new sentence was spoken to her, one she had never learned before, "Button your shoes." The sentence was spoken first without her seeing the teacher's lips. She did not understand it, although the words were in her lip reading vocabulary. The sentence was then spoken while she was watching the teacher's lips, and she at once responded. Then the sentence was spoken again in her ear; she knowing, of course, what was being said, responded at once. Then she was asked to brush her hair, and she responded correctly to that, but when the command "Button your shoes" was repeated in her ear, she did not respond. She had not yet learned to perfectly associate the meaning with those sounds. After a few repetitions she understood readily, and that sentence was added to her hearing vocabulary.

Ruth had a considerable perception of sound, but it was not sufficient to enable her to acquire language or speech up to the age of seven years. If it is remembered what a child of three years with normal hearing can do, it can be seen what a handicap this child had at seven. In four months she has acquired a considerable vocabulary, and uses her knowledge in talking all the time. The amount of residual hearing has a great effect on her speech; it would not have been so clear or good if she had not this perception of sound. It has enabled her to modulate her voice and to speak much better than if she had been born totally deaf.

Virginia: This child is a little older and has a little more hearing than the other, and yet not enough to acquire language in the normal way. (The teacher talked into her ears, and the child repeated the words or answered very clearly. "How old are you?" "I am ten years old." "How tall are you?" "I am four feet eight and a half inches tall." "Where are you going this summer?" "I am going to Mankato." "With whom are you going?" 'With Auntie Madge." "How much do you weigh?" "I weigh seventy-nine pounds." "What is your first name?" "Virginia," etc.)

This child would never have had the speech and language which she now possesses, and would never have acquired an adequate hearing vocabulary, without patient, daily, persistent training.

Alice: Dr. Yankauer had this little girl in his office some years and removed adenoids and tonsils and treated her two or three times a week for two years. Prof. Wright regretted that he was not present to tell about her from his standpoint. She came to Dr. Yankauer without any vocabulary, without any language, without any understanding of words. During the two years she was with him, he and his wife gave her some education for her hearing and some training in the way of a vocabulary, in addition to medical and surgical aid, so that when she finally came to Prof. Wright she had a few detached words.

She was asked in a loud voice near her ear, "What do you study in school?" (The child was very evidently nervous on being called to the platform.) "Geography, history, spelling, drawing, arithmetic, penmanship." "Where are you going this summer?" "I am going to the country." "Where?" "Lime Rock." (The child hesitated and was prompted—"Connecticut"; but corrected the speaker and said "New York.") "Have you any brothers or sisters?" "I have a brother." "Is he older or younger than you?" "He is older." "Have you any sisters?" "Yes, two." "Are they older than you?" "Twelve years old, and one year old." "How old are you?" "I am eleven years old."

In her studies she is about a year behind where she would have been if she had normal hearing. She is very nervous and highstrung, and was very much embarrassed.

Prof. Wright, in conclusion, said: I want you to carry home the idea that if a child has a little perception of sound it should not be neglected. Twenty years ago these children would have been sent into a school for the deaf and dumb, and would have come out deafmutes, with no understanding of speech. There is no reason why these children should be different from any others.

The Problem of the Education of Deafmutes.

HARRIS TAYLOR, LL.D. (by invitation): I was requested to bring some children with me tonight to illustrate what I have to say, as Mr. Wright has done, but I have "The Problem of the Education of Deafmutes," and it is too large a subject to so illustrate. Some phases of the problem might, perhaps, be so demonstrated, but in others nothing would be gained. Douglas Tilden, an artist of more than local reputation, once said that "the congenital deafmute was a born liar and thief, and that he would forget his mother in three days."

There is no question but that the congenital deaf child is exceedingly immature. His notions of property are not in accord with those of the average lawyer or minister, by any means. Whatever he can get hold of and take away with him is his until he is reminded that there is some competition in the matter of possession. Having learned that this assumption has its advantages, he resorts to it in many instances, so that it is not uncommon to see an ignorant, nice, sweet little girl pick up a pencil or something of the kind and vow that she had never done it, and then, if she is caught, she lacks the feeling of humiliation that you or I would have, but in an indifferent manner says: "That is so, I overlooked it," and the incident is closed. Broadly speaking, the uneducated deaf child is a stranger in his own home. He knows nothing of how to communicate with his own people, and they accept him in the beginning as a mystery, and do not attempt to do very much for him. I am glad to say that now, thanks to many persons, such as Dr. Wright and others, there is much literature and information available as to how to treat the deaf child before he is of school age, but parents, as a rule, know nothing of this.

When I began teaching, I spent nearly all my spare time watching the children communicating with each other, to see what they had to say and how they did it. I took the children who had very little knowledge of English, and would communicate with them to the best of my knowledge, and then, for three years I was president of a society for child study, I conducted examinations into the memory of deafmutes prior to the period at which their education began, and I was very much surprised to learn that the child who entered school at eight years, with no previous instruction, could not remember as much of his seventh year as I could of my fourth or fifth, which shows that up to the time that the education began the child had notions of such a hazy nature that they made no impression upon his mind, and after his schooling began they passed from his memory to a large extent.

Perhaps the great difficulty in dealing with deafness is the difficulty of communication. In the beginning I used what is known as the finger alphabet, and quite naturally I thought that when I knew it and the child knew it, the problem was solved, and we could enter into communication at once. I soon found, however, that it did not solve the problem at all. Then I tried gestures, or the sign language; I used these with considerable fluency and could think in them, but here, again, I found that I could not communicate beyond the ideas in the child's mind. In the first place, there was very little communication, and in the second place, there were very few ideas in the child's mind; so that one of the greatest difficulties is to develop the child's mind, and then to place him in communication with others so that he will not be a stranger in his own home. There are several methods of developing the child's mind, all having the same end.

First, the oral method—teaching the child to articulate and understand you from the motion of your lips and face. Next, the finger alphabet—spelling out each word; and, third, a system of gestures, which has been classified until it has been developed into a sign language, all having been used to enter into communication with the child and give him information, food for thought, and place him in communication with the world. Writing, also, is extensively used in this way.

The objectives in the education of the deaf child are to make him a God-fearing, law-abiding and self-supporting citizen. If you succeed in that, you have done fairly well.

Broadly speaking, congenitally deaf children with good minds will complete practically the eighth grade of the public schools in New York in their tenth or eleventh year, the handicap of deafness being that many years.

Then, there are a great many things aside from that. The ability of the child to adjust himself to things that he is out of harmony with. He has to go out of school and maintain a proper relation to his family when they do not understand him—a proper relation to the world around him, where everything he sees is based on hearing and adjusted to the hearing person. We want to make him happy and able to adjust himself to his environment.

Mr. Amateau has brought out some of the difficulties he encounters in helping the deaf to make a living. He has pointed out very clearly the ramifications of business requirements, and the time for all this is so great that the schools do not and cannot yet teach an adequate number of their pupils. It is a big problem to solve. We are trying to help him, and he is trying to help us. There must be a transition period when the school releases the child and before he has obtained the proper apprenticeship to equip him for his position in life.

The better he can communicate with others, the better he will be equipped; therefore, the more he can speak and the more he can read the lips, the more successful he will be, other things being equal.

Bearing this in mind, all the schools lay great emphasis on speech and speech reading, the difference being in the extent to which it is carried out. In my school it is carried out to the exclusion of other methods of communication. In other schools other methods are followed to a greater or less extent; but all recognize the necessity of lip reading and speech as a means of communication with the hearing world, from which he must earn his living.

The problems are, first, to enable the child to become a law-abiding member of society and to support himself, and then to give him as much individual life as possible. They are often able to complete their work and go to college; but, broadly speaking, the congenitally deaf person will never get much beyond the seventh or eighth grade, and the industrial problem is a very great one in their training and should never be lost sight of; they should not only be able to follow a trade, but to understand their relationship with the hearing world, and accommodate themselves to their environment. This last is just as important as to learn a trade.

I cannot go into any details, but we have to develop them mentally and morally, teach them to adjust themselves to the form of society around them, enable them to make a living, and then give them as much of individual life as possible. These represent the real problems in the education of deafmutes.

The Relation of the Hard of Hearing Patient to the Physician.

DR. WENDELL C. PHILLIPS: The third portrait which you see on the wall to the left is that of the physician who gave to Mr. Johnston the keynote which changed his whole manner of life, and who gave to the otologists who are present here tonight a lesson and a sermon which is bound to do us all good. In the strain and stress of our daily work we are too much inclined to look upon these cases as merely cases and not as human beings; and we should enter more into the human side of the question of the relation of the physician to the deaf person rather than to devote our whole efforts to the scientific aspects of the case.

In dealing with the deaf patient, the otologist has to differentiate between the varieties of the deafness in the patients who consult him for treatment. You have tonight heard some of these types mentioned. Inherited deafness, those cases which go from generation to generation, perhaps skipping one generation to again appear in the next; the deafness which comes on at from sixteen to twenty years of age (Mr. Johnston had some aspects of that type of case); invariably, no matter whether treated or not, the hearing of these persons gradually decreases until the time comes when outside help must be obtained for understanding the spoken language. We must recognize that that type of case is not greatly benefited by treatment, and yet there is a way in which the physician may be of real service to the patient.

Another has to do with purulent ear disease, abscesses of the ear in childhood. These cases require prompt and efficient treatment at the time of the primary attack. Children who have repeated recurrent attacks of abscess in the ear are liable to become permanently deaf; but if treated at the time of the first attack they can be cured, though sometimes it takes radical surgical measure to bring about a cure. This type of cases is usually amenable to treatment if taken in time, but if permitted by the parents or family physician to go with the repetition of abscesses in the ear, or without removing the adenoids and tonsils that are often the primary cause of the condition, or without doing a timely mastoid operation that may be necessary for the permanent cure of the case, the hearing will be lost.

Many persons have the impression that if a patient has a mastoid operation it means the destruction of the hearing; but the converse is true. In cases of persistent otorrhea or discharge from the ear, or of recurrent attacks of abscess in the ear that extend beyond the confines of the cavity, operation prevents deafness. In a majority of cases the simple mastoid operation can be done with resulting perfect hearing. It is important that those who do not belong to the medical profession should be aware of this fact. The operation is done in order to save the hearing and to prevent possible dangerous or fatal complications ensuing.

There is still another and a more common type of deafness; but before considering that, I want to refer to another type which has been described by some of the preceding speakers—partial deafness, or deafmutism. From the standpoint of the otologist, there are but few cases of congenital mutism—proportionately, very few cases. It is my impression that very few children are born totally deaf. We know that there are some, but the majority of the cases of even total deafness come on after birth and are due to some inherent dyscrasia or early disease like inherited syphilis or epidemic cerebrospinal meningitis, etc.—they become totally deaf, but are not born so. Very few are born totally deaf. That emphasizes the fact which Dr. Wright spoke of—it is very rare in our clinics to find children that are totally deaf (except those that have had cerebrospinal meningitis). Though the patient may not know it, we know that he can hear a little. Only two days ago I saw just such a case—a child I had seen first at the age of eight months, who had inherited syphilis. The labyrinth and the entire ear apparatus was destroyed. That child has hearing in the other ear—not very much; but he is now four years old, and has not had any training, and has not acquired speech. It could be demonstrated that he has some hearing, and if he could be placed under proper conditions he could be taught as the children seen tonight have been.

The most common of all types of deafness is catarrhal deafness, and I wish to place great emphasis on one particular phase of that type. It is the general impression among the laity, and also among many of the medical profession, that the socalled catarrhal deafness is a disease of middle or advanced life. We see persons with catarrhal deafness begin about thirty or forty-five years of age to lose their hearing; therefore we say that it is a disease of middle or advanced life. This is not true. The basis of catarrhal deafness, in the large majority of cases, is laid during the days of young childhood. Fortunately or unfortunately, the deafness does not commence until later on in life. How do they come? The chronic catarrhal case is deaf because the patient has had repeated attacks of acute catarrhal trouble, and these acute attacks have occurred in childhood.

In my clinics I have taken pains to examine children of ten, twelve and fourteen years of age, and it is very common to find the ear drums permanently retracted and thickened, although the children hear, which shows that they have had repeated attacks of otitis media purulenta. It simply means repeated attacks of obstruction of the eustachian tube, due to colds in the head. If we were to test the hearing of those in this room, one in every three or four would be found to be partially deaf in one ear. I am in that class myself. It is the effect of what you all have had—a cold in the head for two or three days, and then a feeling of stuffiness in the ear, a little bit troublesome, or perhaps a little ringing in the ear. The eustachian tube is temporarily partially closed, a partial vacuum results, and some changes take place in the retained air, and nature in her efforts to overcome the vacuum retracts the drum; then, later, serum is thrown out into the tympanic cavity, and if nothing is done some of that serum remains and undergoes what is called connective tissue formation, and slight adhesions result. That gets well, but another attack comes on, and sometimes there are several of these attacks in the course of the year. If nothing is done for them, the thickening and adhesions increase, and the time comes when the hearing is affected permanently and grows steadily worse. All of this can be prevented, and it is the prevention of catarrhal deafness that I want to emphasize tonight. I want to warn the parents of young children who suffer with these "colds in the head" that something must be done for the children if they are to escape serious results later in life. The treatment is very simple; but patients with this condition should have daily treatment for three or four days in the later stages of these "colds in the head." Catarrhal deafness could be entirely prevented if these patients could receive proper care in early life. It is not a disease of advanced life, but of early childhood.

Dr. Phillips here read a letter received the day before, in which a woman of thirty-five described herself as a working woman whose hearing was gradually becoming impaired, until she was liable to lose her employment, and her troubles were made worse by the fact that others depended upon her for support. She desired expert advice and wished an appointment at an early date, and appealingly hoped that something might be done for her improvement.

On the same day other persons consulted him, suffering with the same type of condition, one of them a young Jewish man who still holds his job. He became an expert before he became deaf, but he is down and out so far as his hearing is concerned. Nothing can be done for his hearing. In addition to his growing catarrhal deafness, he has worked in a noisy place since he was a lad. I can do nothing to help him, and I had to tell him so. I finally got his mind on the subject of artificial aids to hearing, and then in the most tactful manner that I was capable of I brought him around to see that he could not be helped by treatment. These are experiences that come to us every day, and it behooves us as otologists to always bear in mind the human side of the patient. It has been my experience to have many letters like that just read, and the older I grow the more of such letters do I receive. Most of these patients have been in the hands of quacks, and that is a point I always impress upon them. Don't heed the advertisements of aids for the deaf, because not one of them will have any effect. Keep in touch with a conscientious otologist, even though he has no treatment to help. I have many such patients who come to me once in two or three months—some of them because they are downhearted and come for inspiration. They know that I am not going to treat them, but they find it worth while to come for such help as I can give them. This is the relation of the deaf person to the otologist.

Miss Kearns (Principal of Public School 47, School for the Deaf): I was asked by Dr. Hays to come here tonight to say a few words about the vocational work in our day school. It is very important, for we feel the necessity of following our scholars beyond the walls of the school. At first we found it very difficult to get the outside world to accept our children, even though we had done something for them; but at present I am very happy, for people are coming to our school and asking for our children for positions, asking for more children than we have ready. Of course, we have not done anything as big as Mr. Amateau, for we have just begun, but we have been able to keep close to our children. We have had them meet us once a month in social meetings—at first in the evening, and now on Sunday afternoons. They come to us and tell us how they are getting on in the world. In order to get some statistics, we prepared a little card, which reads as follows:

What is your name?
Where do you live?
Where do you work?
(Give the name and address of the firm.)

How much do you earn? (Be honest in answering this.)
How long have you worked in this place?
What kind of work do you do?

These cards are just beginning to come back to us; we have had seventy this week. We do this to keep close to the children.

We found that by sending out to the trades and finding out what they wanted and then preparing the children for the special thing asked for, we could place our pupils more readily. Then we keep in touch with the firm by inquiring how the children are doing. When they reported that the children were slow, we tried to show our children and young people that they had to be quick, and to learn to do things quickly. Then we found out that it was very hard to get them to come early in the morning, and that the day seemed very long to the beginner. Eight weeks ago one of our industrial teachers called the boys together and told them how time cards are kept in business houses, and how it was part of an employe's work to be on time. Now for eight weeks we have had fourteen boys coming in at eight o'clock, signing their time cards, and working earnestly until the bell rings for the regular work of the school day.

We have been very fortunate in our industrial teachers. They have all worked in the trades, and so have a practical knowledge of business. I thought the best way for you to know something about this was to let you see some of my graduates, and let them tell their story. I wrote to these pupils, asking them to write a three-minute composition telling about their work. They are now here to tell you in three minutes how they are getting on in the world. The first girl became deaf when nine years of age, from spinal meningitis. She is now working with a well-known firm of manufacturers of children's clothing. She spoke as follows:

"I was graduated from Public School 47 last January. Soon after, through my teacher, I obtained a position with a firm of manufacturers of children's clothing. I am in the millinery department. I make children's hats. I also make the flowers and the trimmings. There are three of our girls employed there. The forelady says I am a very good worker, and I read the lips very well.

"This dress I am wearing is my graduation dress, which I made myself. I also made and fringed this sash and I made the silk flowers on my hat. Here are some silk violets made by the girls in the school, and the boys made the box to hold them.

"There are different kinds of deaf people in the world. Some do not try; they are afraid. But I mean to succeed. I have my two hands and my two eyes, and I can talk and read the lips, as I am not afraid."

The next girl became deaf when seven years old. She is employed by the Waterman Fountain Pen Company, and is making cases for the pens.

Amelia read in a very good and distinct tone the following letter: "When I was seven years old I lost my hearing. For three years I have been working for the Waterman Fountain Pen Company. It is very interesting, and I have improved very much since I went there. I cover and line the cases for the pens. Here are some I made. I like my work. I can read the lips of the foreman and the other workers, and they understand me when I speak. I have been promoted once and I hope I shall be promoted again."

The next boy is employed by Charles R. Lamb, the artist and designer. He was born deaf. He spoke as follows:

"I was graduated from Public School 47 in 1912. The following fall I obtained employment as draughtsman in the studio of Charles R. Lamb. I take all orders from my employer's lips and find no trouble in doing so. I also converse with the other people in the studio. I have had a raise in salary, and I am happy in my work."

The next boy will be graduated next February. He has not quite finished his course. He has done some very good work in lettering, and we feel that we can easily place him in an architect's office. The young man spoke very clearly as follows: "I became a pupil of the school for the deaf about four years ago, and since that time I have made great progress in industrial design, particularly in learning the different styles of letters and the ratio upon which they are built, and also the process of laying gold leaf and other processes employed in sign writing. When I graduate I hope to enter an architect's office, where I will learn the trade of designer. These are some of my signs. This one shows how I use the gold leaf. This one shows another process of putting on a red background. This is some of the work which I have made outside of the school. It is also the number of our school. Some of the hearing schools down town are going to have a Shakespeare pageant, and I am making some signs to show the road to Shakespeare's country. This is 'Warwick, 3 M.' It is not finished, but I will finish it now."

These compositions are all of the children's own writing. I have not seen any of them. The last boy who will speak to you has had a chance to go to high school; two boys have finished two years in high school. One boy now in the Stuyvesant High School has done very well. At the end of his first year he stood highest in his class. Another boy is in the East Orange High School, and a third is in the high school in Bloomfield, New Jersey.

The boy in the East Orange High School also addressed the audience very clearly and distinctly. He said: "This reminds me of an incident in my life before I came to the school for the deaf. I was in the grammar school, and at that time I had a very imperfect speech. I was anxious to overcome that. Among the advertisements I learned of an article which advertised that it could make your voice good, and was used by singers and speakers. I thought it would improve my speech, so I went and bought it. It was Lymole Trochees. I ate a whole box, but they didn't help my speech.

"I went to the grammar school before I came to the public school to learn. I did not learn much there. Things were not clear to me. I did not know enough to help myself. I could not hear clearly, and I became confused. The day I entered the school for the deaf is a milestone in my life; the door to a new world was opened to me; everything was made clear to me, and, best of all, I was learning to speak right. After a busy and delightful three years in that school I graduated. I was at first reluctant to enter high school, for I remembered my grammar school experience. Now, after two years, I am finishing half of my course, and in that time I have made a record of which I am not ashamed, and I hope to attain a college scholarship.

"I do not hesitate to take up the different activities of school life. I am a member of the track team and have made a name for myself. I have here one of the many tokens that I have won in the past two years. Among the trophies that I have won are two cups. The last one that I won was third prize in the one-mile championship of America, about three weeks ago.

"Besides that, I am now a candidate for the position of editor-in-chief of the high school magazine. I have written for that publication several times. I am also to take part in a play to be given later this month. My principal, Miss Kearns, and the teachers, are steadfast friends and are among the best I have. Through them, and the oral method of teaching the deaf, I have been able to make a success during the last few years. Through these teachers I have learned to say 'Where there is a will there is a way,' and subsequent events have made me a firm believer in that saying. As you have seen, I am not the only one who has been helped by the oral method of teaching the deaf. I believe the lip reading method is the best in the world for the deaf."

MISS J. D. CLARK (Assistant Principal, New York School for the Hard of Hearing): I feel highly honored to be asked to take part in this meeting tonight, and I am very glad to tell what lip reading means to the adult hard of hearing. It is a joy and a privilege to teach lip reading because of the difference that it makes in the lives of deaf people.

I consider that the spiritual factor of lip reading is quite as important as the mental and physical. If the spirit is not willing the flesh cannot do much.

Most of the people who come to us are eager to learn; eager to be aroused from the depression and mental sluggishness into which deafness has caused them to sink, and it is wonderful to see the difference that lip reading makes in them in a short time. The very expression of their faces changes, and from hopeless, discouraged people they grow to be hopeful, courageous people. Seeing others bravely fighting the same handicap inspires them to do likewise.

But sometimes people come to us not because they want to come, but because their families make them come. They have the attitude of "I can't learn. It is too hard." The first thing to be done is to change that attitude; to make them see that they can learn. It is seldom that this cannot be done.

I have in mind the case of Miss B., who took lip reading several years ago. She came because her mother made her come, she, herself, being opposed to the idea of lip reading. In a very short time her attitude was completely reversed, and when she left the school she was one of our best lip readers. But more than that, she had overcome her mental depression. When she went away she said to me: "If only one person out of seventy-five learned to read the lips, you are doing missionary work because of the cheer you bring into the lives of the deaf."

This pupil later took the normal course and is now teaching lip reading herself. Many of our pupils do this, seemingly in an effort to help others as they have been helped, as well as to provide for themselves a means of self-expression and support.

Mrs. T. is one who took this course, and the change it has made in her was evident to a relative of mine, who knew her when she first became deaf. This relative went to call on her at that time and found her quite hopeless to talk to, as she would not even look at the speaker. Last year my relative saw her again and found her full of life and enthusiasm for her work; the kind of person it is a pleasure to be with.

It was not so very long ago that many aurists opposed lip reading for the partially deaf, on the ground that the hearing would deteriorate if too much dependence was put upon the eyes. Most of them have changed their opinion, I believe, although there are still some who hold to this view. This has not been the result in my case, for I knew that I am not any deafer than I was when I began the study of lip reading twelve years ago. The fact that I use what hearing I have probably has something to do with this. I always watch the lips, but I hear what I can at the same time.

We always instruct our pupils to do this—to make their eyes and ears cooperate, so that lip reading is an aid to hearing, not a substitute for it, and the strain on the ears is relieved just enough to be good for them.

We have had aurists send us patients who were very nervous from trying to hear, and the lip reading relieves this nervous tension and generally improves the person's health.

Mrs. M. writes: "Somehow, since taking lip reading I've calmed down. When people talk to me my face doesn't twitch nervously. I hardly ever get flustered. I feel more now as if I could hold my own than I ever did before. Not that I always know what folks are talking about, but I am better able than before to get along."

Miss C., a teacher of a large kindergarten, writes: "When I became deaf it was no easy task to keep on teaching. The lip reading has made it much easier by relieving the strain."

Lip reading is harder for some than for others to learn, but almost any one can acquire it if he or she will keep at it long enough, and it is well worth the effort. While it is not the same as two good ears, it is a very great help, and armed with a knowledge of lip reading, one still has a fair chance among men to work and play.

Dr. Walter Lester Carr said that the education of the deaf should include sociologic and economic interests, to enable them to make their way in competition with normal people, and to give them interest outside of themselves.

He confirmed what Dr. Phillips had said in regard to the small number of children who are congenitally deaf. This he had verified by his observation of many children. Very few are congenitally deaf, although they are usually so classed and accepted. From the pediatric standpoint, he agreed with what Dr. Phillips had said in regard to the prevention of deafness. A great deal can be done toward preventing deafness in children who have suffered from scarlet fever, measles, influenza, etc.; also in children with diseased tonsils, adenoids and nasal deviations. These children are almost invariably seen first by the pediatrician, and careful, simple treatment at the proper time would prevent many cases of deafness. There is no group of cases that offers better opportunity for a physician to care for a child and prevent future injury.

Mr. Amateau said that it was all very well to show what wonderful results were accomplished by the oral method of teaching the deaf. He said he recognized that the oral method enables the deaf to speak and receive orders from their employers and foremen and others who must communicate with them by word of mouth, and that such work as had been demonstrated tonight had much value as far as industrial needs are concerned.

It was all very well to point to these children who had some hearing and who had become deaf after a few years, but he would like to see a person who had been born deaf or had become deaf very early, and then acquired speech in a school for deafmutes, understand from the floor from the movement of his lips what he was saying while standing upon the stage. If he could see this done, he would become a convert to the exclusive oral method of teaching, but he did not believe it possible.

He had tried it in a number of cases. He had been with Mr. Wright's graduates and with Miss Kearns' graduates, and had placed a number of Dr. Taylor's graduates, all of whom were taught exclusively by the oral method. He further said that when you get heart to heart with them, they agree that it is the very hardest sort of strain upon the eyes and that it is impossible to read the lips under such conditions for more than five minutes; that they must depend on some larger means of communication; so that while he believes that speech reading and articulating are industrial assets, yet if the deaf man is to enjoy religious and other lectures and social meetings (which he must have) he must learn to depend upon something other than mere lip reading. People should not gain the impression that lip reading meets all the requirements of the deaf. Any number of deaf people will tell you that they cannot depend upon it, and that they depend more upon the sign language; witness the fact that a majority, if not all the graduates of exclusive oral schools, communicate by signs with their fellow handicapped. Speech reading and articulating should be taught as much as possible for the opening of the mind, but there are boys and girls who are not bright enough to acquire an education through the oral method exclusively, and they should not be taught just lip reading and articulating, as mere parrots, and sent out into the world to make their living. He had had to take that kind of product from the schools and place it and try to make something of it. It is an injustice to the boy and girl, and an injustice to society. Such persons should be taught the sign language also, and educated through the sign language, when the oral method failed.

The object is to make them useful and intelligent citizens, and no one can become such unless he understands what the world is and what his privileges and duties to society and himself consist of. It is not only the means of communication that a deaf boy needs, but also an education, so that when he does talk, he should know what he is talking about.

PROF. WRIGHT said that he had not expected to say anything further, but that he must make some response to what Mr. Amateau had said. He felt that he was fairly familiar with the English language, and yet he had not understood more than one-third of what Mr. Amateau had said when reading his paper on account of his foreign accent. Now, a lip reader who has been accustomed to perfectly normal English speech would find it very difficult indeed to understand him, but he could become accustomed to it, and in the course of time, by associating with him for an hour or so a day, he could learn to understand him.

Mr. Amateau's speech was exceedingly difficult to understand, even for a hearing person.

In regard to signs or oral teaching, Mr. Wright said that he differed from Mr. Amateau. It was possible to learn the sign language so that it could be used with a reasonable amount of facility in from two to twelve months. It was possible to learn the finger alphabet in six or eight weeks, and use it with fair facility. It was not possible to learn to speak or to read the lips in less than from five to ten years and to use speech as a natural mode of expression, a living vernacular and not a translation. It is an easier thing to educate a child with signs and finger spelling; it is an easier means of communication, and if the world as a world used signs and finger spelling, it would be well to employ that method generally for the education of the deaf. But it is not a universal means of communication, and if each deaf child is to reach his own individual highest efficiency in speech and speech reading, he has to have it all through his educational period.

Personally, he had no more objection to the sign language than to French or German. His objection to its use was during the educational period, when the mental habits of the child are formed. After they have acquired the ability to express themselves, there was no objection to their learning the sign language and finger spelling. Both have their place, but not in the educational period. It destroys the ability to learn speech in its highest possibility. One cannot make a Paderewski out of every person that plays the piano, nor can you make a brilliant speaker or lip reader of every deaf child, but you can give each one the highest efficiency he is capable of. You cannot do this so long as manual methods of education enter into their early training. If, when he is eighteen or thereabouts, he wishes to learn to use and understand the sign language and finger spelling for the sake of attending lectures and to associate with deaf persons whose lips he cannot read and who cannot understand him, there can be no possible objection.

Prof. Wright, referring to Mr. Amateau's remarks, said that he could talk to scores under his personal teaching by the oral method, and could talk to them for an hour. Not long since he had made a speech at a dinner given by the graduates and pupils of the school, and they cheered at the right place and gave him to understand afterward that they approved or disapproved, as the case might be, what had been said, and one of them, who was born totally and congenitally deaf, and had never heard a sound in her life, wrote a correct verbatim report of what had been said.

Dr. Harris Taylor said that he felt as Mr. Wright did in regard to the sign language and finger spelling, or the manual alphabet. His own experience dated back into the last century. For two and a half years he taught the deaf by means of the sign language, interspersing it with the use of language written or spelled, and by this method allowed eight or ten pupils who had a residuum of hearing to become deafmutes.

It was only with some pressure that the oral method was introduced into the Texas school. He had asked for it to be used in two classes, though he did not expect anything remarkable from it. He became convinced, however, that for school room purposes, spoken and written and spelled English should be used, and for a period of four years he followed that method, using no gestures at all.

Then for seven years he tried the oral method in the school room, using speech and lip reading and writing as in actual life, without the use of finger spelling or signs.

Then he took charge of a school, directing it in two divisions, part oral and part manual, supervising that work for seven years, and after that for another seven years he supervised work in a school where the pupils were taught by the oral method alone.

As an educational measure, if he thought that if the child sacrificed anything at all in the use of language as taught by the oral method, he would not hesitate to use the sign language, but it is not necessary. In the best schools of today, and those which carry the children highest in a given time, the English language only is used in the school room.

Another point of importance was that in the formative years of the deaf child he should be taught to use and observe the lips for talking to those around him. He is not educated to be a leader in a deaf and dumb community, and every one that can be educated away from a deaf and dumb community represents good service rendered.

Dr. Taylor said he thanked the Lord that he had been able to contribute something to society by taking deaf persons and educating them to lead a normal life with other persons.

When it comes to the pleasures of life, if the deaf find any help or pleasure in the sign language after they have acquired the habit of speech and speech reading, let them have it. If they find it in finger spelling, let them have it. They should not be deprived of any joy or advantage that can be found in the sign language or any other way. If any group of adult deaf persons wished him to address them in the sign language. he would be glad to do so, but as an educational factor he believed that the oral method meets the conditions best for the comfort and enjoyment of life. During the formative years in school they need the English language to qualify them for this and for mingling with those that they must make their living from in after years.

Dr. Phillips, referring to the remarks of Dr. Taylor, said that they constituted a very strong argument in favor of the speech method of teaching the deaf rather than by signs. From observation of the young people who had spoken tonight, he could see also that they think more quickly than those who use the sign language. In his opinion, there was no question but that it was the proper method of teaching the deaf. One point, however, had been neglected by all the speakers. None of them had said anything about how early in life these children should commence to receive their instruction. He recalled that in his undergraduate course Dr. Wm. H. Thompson had laid much emphasis upon the degree of education gained by the normal child under three years of age in comparison with what he learned later—motion, speech, emotions, ideas, etc., all before the age of three. If he is deprived of hearing and his education for speech is delayed until he is six years old, he has lost much valuable time. In his judgment, children that are deaf ought to be brought under special instruction earlier.

The work for the deaf in the public schools is one that New York has reason to be proud of, and ought to receive the support and encouragement of the public, and a better building should be provided for it. The public knows very little about what is being done for these children, and more publicity should be given to it.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of March 21, 1916.

DR. OTIS H. MACLAY, THE PRESIDENT, IN THE CHAIR.

DR. H. B. YOUNG, of Burlington, Iowa, by invitation, presented three cases.

Congenital Absence of Soft Palate.

DR. YOUNG said that all the members had probably seen cases of congenital absence of the soft palate. The problem of repair by operation or prothesis was before him in this case, and he had had an expression of opinion from the members.

Oralism for the Deaf.

The second and third cases (two adults) furnished an excellent demonstration of the finished product in oralism. Case 2 and his colleague (Case 3) are the managing officers in Chicago of the National Fraternal Society for the Deaf. Both are equally capable business men. Dr. Young showed them in order to demonstrate what actually takes place in the matter of development, education and social uplift of the totally deaf by the different means of education. It has been said by some that the speaker is opposed to oralism. He is not opposed to it; but he would not want the profession to believe that in pure oralism we have a solution of the problem of what is to become of the incurably deaf. (The first of these two patients—semideaf and semimute—then gave the history of his ear condition and education, which demonstrated that pure oralism does not do what is claimed for it in adult life.) The second of these patients could read the lips only very imperfectly, and depended on signs.

The main point of Dr. Young's contention is that the propaganda of oralism approaches charlatanry; and that as otologists it is our duty to tell these patients what may be done, and the limitations of oralism.

The first man said that the consensus of opinion of the educated deaf all over the country is in favor of the combined method of education, and utterly opposed to the use of the oral method exclusively.

Dr. Young then called attention to the difference in the voices of these men.

DISCUSSION.

Dr. Kenyon asked if there had been any attempt made to change the tone of the voice, to which the first patient replied that he was once sent to a teacher who tried to do this, but not with very much success.

Dr. Shambaugh said that the great object of oralism was to allow these patients to talk with others who were not deaf and did not use signs.

Dr. Young replied that we all unconsciously use signs in talking. The pure sign language is universal, and everybody can use it. This is carried out by the "movies."

Dr. Kenyon asked if Dr. Young were not minimizing the advantages of the oral method.

Dr. Young replied that he did not intend to do so, but thought that it had not benefited the deaf very much; that is, not so much as was expected.

Dr. Kenyon asked if it was not dangerous to put forward the sign method too prominently. Does it not lead to laziness on the part of patients? Would not the tendency be to give up the harder method in favor of the easier?

Dr. Young said that the majority who have not investigated the sign language do not understand it. It is a pictured idea.

Dr. Arthur M. Corwin asked if it did not make a great deal of difference, to even the patient, in a mixed crowd, as to who is speaking. To which the semideaf patient replied, that if people did not try to make it easy he could get along very well. The exaggeration of movements of the lips confused the lip readers very much.

Dr. Kenyon asked if the totally deaf man who was more dependent on signs could do as well as the lip reader, to which Dr. Young replied in the negative. He had tried to learn lip reading, however, and could not master it; so what was there to do about it? The combined method was the most useful.

Dr. J. Holinger asked how long these two men had had lessons from competent teachers who had had a long experience, and could show other results. About eighteen years ago he had showed before the Chicago Medical Society two men, both of whom became deaf much earlier than either of those shown by Dr. Young, one at four, and the other at six. It is well known that it is much more difficult at that time to preserve speech. Both of these patients had very competent training. One spoke German and learned English after he was full-grown. The other one spoke German and French, when he came to this country and mastered English without a teacher. He is a typesetter, and acquired a correct pronunciation and spelling. Very much depends upon competent teaching and long enough training at a time when the patients are really receptive.

Dr. Young, in answer to Dr. Holinger, said that in contrast to his two cases, Mr. and Mrs. Bayliss, who are teachers in the deaf school at Belleville, Ontario, are examples. Mr. Bayliss lost his hearing at the age of seventeen. He naturally speaks very well. His wife lost her hearing before seven, and speaks poorly. Mr. Bayliss cannot read the lips at all, with all the effort he has put on it. His wife is one of the most expert in this country at lip reading. The difficulty is not in speech, but in training the eyes to interpret the signs made by the lips in speech.

Case of Syphilis and Carcinoma, Possibly Both.

Dr. George A. Torrison presented a patient, Mr. W., aged fifty years. This man's wife died in 1906, probably of tuberculosis. Father died at the age of ninety, of old age. Mother at eighty, of old age. One sister died at twenty-nine, probably of tuberculosis; one died in childbirth, at thirty; one sister living, at thirty-four. He has two brothers living, aged forty and thirty-six, both healthy. Seven brothers and sisters died in early childhood.

The patient had gonorrhea at the age of twenty; also had a sore on the penis, with adenitis. No venereal symptoms for past thirty years. Has had slight attacks of muscular rheumatism from time to time for several years. Has had no attacks for last four years.

The present trouble began in September, 1915, with difficulty in swallowing. He felt as though something stuck in his throat, which he could not get up or down. This difficulty in swallowing gradually increased until he was unable to swallow solid food. He has never complained of actual pain, but swallowing became more and more difficult, until sustenance was limited to liquid food. He has lost about ten pounds in weight.

In December, 1915, he was operated for fistula in ano; since then there has been more or less constant discharge.

When Dr. Torrison first saw the patient, last January—about two months ago—he could only swallow liquids, but had no pain. The voice was unaffected. The epiglottis was about all that he could see, on examination; it was one nodular mass. A Wassermann was made, which was slightly positive, and the patient was immediately started on antisyphilitic treatment. He has taken about thirty mercurial inunctions, combined with iodid of potassium, three times a day; also injections of cacodylate of sodium, the first of three grians, and the last four injections, twice a week, of five grains each. Shortly after beginning this antisyphilitic treatment the appearance of the epiglottis changed. From being one mass, it became a cleft affair. Swallowing has been easier, so that now he is able to swallow solid food—with some little difficulty, however. There has been some glandular involvement on both sides of the neck. had consulted the speaker on account of obstructed nasal res- The glands on the right side have disappeared under the antisyphilitic treatment. There are still one or two on the left side.

Dr. Torrison brought the patient to the society to get some light on the diagnosis. He has thought that the diagnosis lay between syphilis and carcinoma, possibly a combination of the two. Although there appears to be some tubercular history in his family, he thinks tuberculosis can be excluded, because there are no lung findings, no elevation of temperature, no pain on swallowing, and if it had been tuberculosis, he thinks the process would have progressed much more rapidly than it has under the large doses of iodid of potassium he has been taking. He does think there is a syphilitic element in the case, but whether it is purely specific or a combination of syphilis and carcinoma, he could not feel sure. It has yielded very slowly to treatment. He has been under treatment for two months, and a week ago he began to complain of loss of appetite, so the iodid of potassium was stopped, and the appetite has improved. Since stopping the iodid of potassium he has gained two or three pounds in weight. He had an accident while under treatment, being run over by a motor truck, and was in bed for three weeks, and has not felt quite so well since that time.

Adhesions of Posterior Pillars to Posterior Wall of Pharynx.

Dr. George W. Boot showed a young man who had adhesion of the posterior pillars to the posterior wall of the pharynx. He piration, which was due to a defective septum, and had no connection with the condition in the throat. The patient knows nothing of when the throat trouble began, but gives a history of having had scarlet fever when a child. The speaker thought the condition was undoubtedly the result of an ulceration of the throat from the scarlet fever. There was only a narrow opening, about one and one-half centimeters long, and one-half centimeter wide in the midline. There is evidently a band underneath, which is adherent all the way across.

Dr. Boot intends to separate each pillar from the pharyngeal wall and put a mattress suture through, bring the ends out and tie them, so as to fold the pillar on itself, and let the pharyngeal wound heal by granulation. The pillar would be covered on both sides by mucous membrane.

DISCUSSION.

Dr. C. M. Robertson said he would like to see how much contraction occurred after the operation described by Dr. Boot. He thought it probable that the operation would have to be repeated.

Case of Decompression of the Hypophysis.

Dr. Otto J. Stein said that the patient was operated on about three weeks ago by the transsphenoidal-nasal route, the operation being done upon his sella. He was operated for blindness, which had been present for five years. He was blind even to light. X-ray showed tumor in the neighborhood of the hypophysis, causing pressure. The patient did not present any of the symptoms of hypophyseal disease. The only thing that indicated involvement of the hypophysis was the blindness, aside from the condition shown by the X-ray picture, which was a very good one. The speaker's principal reason for bringing the patient was to demonstrate what the rhinologist can really do by a decompression operation. Of course, decompression operations can be done otherwise than inside the nose, but the rhinologist has in his field an avenue of approach that is, in the speaker's opinion, even superior in its simplicity, in its safety and in its final results, than the general surgeon has, by the frontal or temporal, or through the oral transsphenoidal route. Then, too, the rhinologist has a technic at his fingers' ends that does away with many of the disagreeable features of the transsphenoidal-nasal route that the general surgeon employs.

In following Hirsch's technic, a simple submucous resection of just enough septum to reach the ostium of the sphenoid, and removing only one middle turbinate, are necessary. The speaker has done that in all of his cases, but only because all of them have had rather small nostrils. He thinks that in people with very large nostrils that would not be necessary even. But you do require a little bit more room than you get in an ordinary nose without removing the middle turbinate. That, of course, means nothing at all to the patient. We leave no disagreeable result; no dryness; no disfigurement; no loss of the sense of smell or discharge or other deformity in the patient's head by such a technic, and still have all the room needed.

This patient was operated under scopolamin-cocain anesthesia, with the patient perfectly conscious and talking all the time. He did not have any pain to amount to anything; even the entrance into the cranial cavity did not cause much pain. The result is very gratifying. The man can see very well the ordinary objects about him. He can also distinguish colors. He is able to get around very well alone.

The pathologic findings will be reported at a later meeting.

DISCUSSION.

DR. J. R. FLETCHER had seen the operation on Dr. Stein's patient, and was very much pleased with the brilliancy of the work. Dr. Stein did a submucous, removed a minimum of the septum and one middle turbinate, took down the anterior wall of the sphenoid, and then the floor of the sella. It could all be seen very distinctly by the speaker. There was no blood to speak of. The pulsation of the hypophysis could be seen. He was impressed with the ease of approach in an area which until recently had seemed unapproachable. And yet there was all the room desired without cutting of the lip or nose.

Dr. Fletcher referred to a case reported before the society several years ago by Dr. Kanavel. However, he thinks the operation described by Dr. Stein is infinitely superior to any other for this work.

Paper: Intramural Malignant Tumor of the Lateral Wall of the Nasopharynx.*

By Edward P. Norcross, M. D.

DISCUSSION.

Dr. Otto J. Stein wanted to emphasize some of the points brought out by Dr. Norcross, in regard to malignancy in the pharyngeal space, because it has a direct bearing on the subject of malignancy of the postnasal space. He saw a patient some years ago (the wife of a physician), who referred the pain to the left tonsil. On very close inspection the case did not show anything at all. She had been operated years before, and there was some scar tissue very deep in the fossæ. There was no evidence of irritation. Dr. Stein told the patient and her husband frankly that he did not know that there was anything the matter with the left tonsil. He made an application of Lugol's solution, which had a very beneficial effect. She returned in a week or so for another application, because she said it helped her so much, and she kept coming back. The speaker told her there was no sense in her coming, because he could not see what effect the applications would have. He decided it was a case of hysteria. She finally fell into the hands of a neurologist, who also said it was a typical case of hysteria, and Dr. Stein was glad to have his diagnosis confirmed. Shortly thereafter she returned to Dr. Stein, because she said she did not think the electric treatments given her by Dr. Grinker were benefiting her, and that Dr. Stein's treatments did her more good. The topical applications were continued, but she came at greater intervals and was suffering more. He did not see her for a long time, and one day a colleague told him that she had been operated on for carcinoma of the esophagus. Dr. Stein then realized that probably he had a case of carcinoma of the lower pharynx to deal with, which he had not suspected. This was in the days before the common use of direct laryngoscopy. This ulcer was discovered in the neighborhood of the cricoid. She had no trouble in swallowing, early in the course of the trouble. It was all referred to a pain in the left tonsil. Of course, later on the evidences of ulceration and breaking down of tissue became more manifest, and the diagnosis was easy.

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*See page 967.

This case taught the speaker a lesson about referred pain, and not long after that he had another similar case. This time, instead of merely looking at the nasopharynx, he looked at the lower pharynx also, and found the same condition of infiltration in the lower pharynx, beginning with the esophagus, which turned out to be malignant, and later was operated on.

Dr. Arthur M. Corwin said that all of this goes to show that all of us, who are used to seeing many cases of a given sort, are apt to become contracted in our way of viewing them. We specialists look through a narrow hole at the pathologic world, unless we have an invariable system, even when busy, of making a complete examination of each patient, from head to foot. In saying this he was soliloquizing. Most of us who have been in practice for a quarter of a century can remember just such cases, where we have overlooked one thing in centering our vision upon another, failing to take a general account of stock.

Dr. George M. McBean said the case cited by Dr. Stein reminded him of one he saw only the week previous. Dr. Lewy saw the patient with him. This patient complained of throat trouble for two years—the principal complaint being of pain in the throat on swallowing, extending up to the ears. She went to a physician, who diagnosed it as rheumatism of the pharynx. She had some carious teeth, which were extracted, but still the rheumatism continued. So she went to another physician, who found she had a retroverted uterus. A ventrofixation was done. The uterus fell back to its original position two weeks later. She had a third operation, and had a normal uterus removed. That did not improve the rheumatism of the pharynx at all. Then she hunted up a foot specialist, who put some braces under her arches. She wore those for some time, but still continued to have rheumatism of the pharynx—in fact, this condition was increased. She declined a tonsillectomy, which had been advised. Last January, about twenty months after the onset of the trouble, she spit up some blood, and so they decided she had tuberculosis, and put her out in the back yard, in a temperature somewhat below zero, and gave her an electric pad to keep warm. That did not cure her either. Then they started in with injections of tuberculin. Two other kinds of vaccines were made, to be given on alternate days, and she was also having internal medicine—large doses of the salicylates. She lost fifty-five pounds. This woman was forty-three years of age. When she came to see Dr. McBean she was just able to hobble in, and had a marked cachexia. She had a very beautiful epithelioma just below the arytenoid cartilages, which had ulcerated, with a slough on the top of it. He recommended a surgeon for a gastrostomy, and also suggested the use of radium. The internal medication was stopped, and the patient has gained a little in weight. This growth was in the hypopharynx instead of the epipharynx, which would have been more appropriate to the discussion of Dr. Norcross' paper, but still he thought the case of interest.

(On April 19, 1916, a section of the growth was examined microscopically and pronounced squamous celled carcinoma.)

Two Wassermann tests were negative. The sputum examination for tuberculosis was negative. An attempt was made to put the esophagoscope, but was unsuccessful, as the growth had invaded the esophagus. There is no intralaryngeal involvement manifest (April 19, 1916).

Dr. Charles M. Robertson did not know that cases of malignancy of the nasopharynx were so rare as Dr. Norcross said. The paper confined the subject to growths of the pharynx itself, without considering the sphenoid sinus, and so he supposed that growths on the posterior septum would be barred. The growths that are common in the nasopharynx are fibromas, and whether fibromas are malignant or not, he did not know, but most of those he has seen were. He has seen quite a number of growths in the throat, in the lateral wall, but nearly all of them were extensions from the sphenoidal sinus. He has had one classical case that would come under this head, which Dr. Sonnenschein also saw, which was a fibrocarcinoma, with a recurrence in the larynx. Laryngeal fissure and an exenteration of one side of the larynx produced a cure. This case is old enough now to warrant saying that it is probably beyond recurrence, as it was operated upon six or seven years ago.

He has had two cases within the month of tumors of the nasopharynx that he could not say were malignant, but he thought they were. As yet he has not received the report. One case sprang from the roof of the pharynx. It was about the size of a medium sized potato, in which the soft palate was pushed almost halfway to the incisor teeth. The patient was a boy of ten or eleven years. He thought examination would prove it to be a fibrocarcinoma or a myxosarcoma.

In the other case the attachment of the tumor was immediately behind the septum, in the nasopharynx.

He did not understand why there should be very many tumors in the side wall of the pharynx unless the tonsil was involved. There is nothing on the side wall of the pharynx except the superior constrictor muscle and a little lymphoid tissue. He had exhibited to the members several years ago a lymphosarcoma that commenced in the tonsil, and rapidly involved the side wall of the pharynx, including the lymphoid tissue around the eustachian tube. That kind of case is not particularly rare. The tumor was encysted. Lymphosarcomas are quite often encysted. That patient was referred to him by a general surgeon, who did not like the looks of it, and so sent it to the speaker. He imagined the surgeon was afraid the patient would die under the operation. In taking a piece of the tumor out for microscopic examination, he found the mucous membrane seemed to be distinct, and the tumor absolutely yellow in color, so that when the time to operate arrived, all that was necessary was to slit the mucous membrane and shell the tumor out with the finger. It only took a moment or two. The tumor extended up between the mouth of the eustachian tube on the side wall, and involved the tonsil, not as a tumor of the tonsil, but included it. The tonsil seemed to be taken in by the tumor. A difficulty that arose during operation, however, was the fact that the tumor was so large that he could not get it out of the mouth after freeing it from its attachment, and he had to work it out with both hands.

Dr. Robertson thinks that probably nearly all of these tumor growths in the nasopharynx are secondary. He remembered one of the first cases a friend of his ever had, which was one of these broad-based fibrosarcomas, which was removed with a snare. One thing he wanted to point out, namely, in the removal of a growth like that in the nasopharynx, it is best done by the ecraseur, which is put on and the tumor cut off slowly, several hours being taken for the removal of the growth. There is nearly always a terrific hemorrhage, which may be obviated in this way. There is a very large blood supply to these parts, and they are spaces in which you cannot very well apply a pack, because of the situation in the throat.

Dr. George E. Shambaugh has seen one case that is quite like the one reported by Dr. Norcross. The patient was a medical student whose complaint was disturbance of hearing. The ear trouble was caused by an occlusion of the pharyngeal orifice of the eustachian tube, due to the tumor in the nasopharynx. The ear symptoms continued to be one of his chief annoyances until the end, and required frequent openings of the drum membrane in order to relieve sensations of pressure, caused by rarefaction of air in the tympanum.

Dr. Norcross, in closing the discussion, said there were many reports of sarcomas in this space, but he had confined his paper to carcinoma. What he wished to call attention to was that often there was very little to be seen when one examined the postnasal space, and the symptoms so frequently complained of were those either of deafness or enlarged glands in the neck, or of neuralgia.

Paper: A Study of the Physicomechanic Function of the Faucial Tonsil.*

By Elmer L. Kenyon, M. D., and W. T. Kradwell, M. D.

DISCUSSION.

Dr. Otto J. Stein said, as he understood the paper, it seems almost absolutely essential to the function of the palatoglossus muscle to have a tonsil back of it, and it is almost as important to have a tonsil in front of the palatopharyngeus muscle. He thought the diagrams and paper very instructive, bringing out very many interesting points, but if it is a fact that the tonsil is necessary for the proper function and activity of these two muscles, how does the doctor reconcile the fact that in cases where you have a normal retrogression or atrophy of the tonsil, where no vestige of the gland is seen, you can have such a perfect-looking, anatomically constructed throat? These cases are seen repeatedly. He could not see how that would apply to the absolute necessity of a tonsil leaning up against these two muscles. There is no doubt but what the operative interference produces changes spoken of by Dr. Kenyon. Of course, we all know that. But we should compare the results following operative work with the condition that would obtain without operation. From a practical standpoint, a great deal more is gained, from the operative viewpoint, based upon the indications for operation, than from the results of operative work—that is, minor work. Weighing the matter this way, it seems to him the objections are very slight.

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*See page 862.

Dr. George E. Shambaugh has been impressed with the marked alterations, as the result of scar formations, which occasionally follow the enucleation of the tonsils, but which produce no disturbance of the voice. The disturbance of the voice seems to be a very unusual sequel to such alterations. In one case with very large upper lobes, which extended upward between the folds of the soft palate, and were adherent because of recurring infection, Dr. Shambaugh experienced a great deal of difficulty in getting the tonsils removed. The patient suffered for some weeks from a paralysis of the soft palate, which caused difficulty not only in speaking, but in swallowing food and water, the liquids passing up into the nose. Even in this case the end results were quite satisfactory, with, in fact, no permanent difficulty whatever.

Dr. Shambaugh has always been very conservative in operating upon singers, and has refrained from operating in these cases except under rather urgent indications. In the cases of this sort which he has operated he has never had anything but a satisfactory result. Still, one never knows when the tonsils are enucleated just how much alteration may follow from scar formations.

Dr. Kenyon pointed out the objections to a tonsil enucleation which rest upon the alterations in the pharynx following this procedure, but he does not suggest a suitable substitute for the operation. As a matter of fact, tonsil enucleation is not undertaken lightly, but is only done in cases where there exists ample justification, because of the menace from tonsil infection. Where these indications exist, the objections to the operation appear very insignificant as compared with the danger of leaving the tonsils alone. Any operation short of enucleation has so far proved entirely unsatisfactory. Cutting off the tonsils, or attempts to cut them out, leaving the capsule, are followed too frequently by an increase in the trouble to justify, as yet, the employment of such methods.

Dr. Arthur M. Corwin said that the spirit of Dr. Kenyon's paper was better than the paper. He did not wish to be misunderstood by anyone when he said that. He meant that the careful, thoughtful approach of a subject like this, in the face of ten thousand or one hundred thousand tonsillectomies, with a view to determining the different phases of the end results, is the kind of work that we want to see. From that standpoint, he thought it was a good paper. He thought that possibly Dr. Kenyon's results or final statements were a little more dogmatic, perhaps, or a little more final, than he would wish eventually to make them. In other words, he felt that Dr. Kenyon was not through with his investigation of the subject, and was not yet ready to say that the tonsil is absolutely necessary for good physiologic and good anatomic relationship, so far as these pillars are concerned. The essayist's conclusions would lead him to rather an equivocal position. The classification struck him as far-fetched, and the estimates based too much on guess-work to make the conclusions valuable.

Dr. Corwin has operated on a good many cases of tonsils by various methods. He has not been able to study the end results as he and others would like to study them. His patients have not come back to him complaining of loss of voice or disturbed function, nasal voice, faulty singing, faulty speech. He has not seen them, and he thought that was probably the experience of most of the members. It would be a very nice thing to corral these patients and study them as Dr. Kenyon has begun to attempt to study his cases.

While Dr. Corwin has not seen any cases of impaired voice from a tonsillectomy, either among his own or following operations by colleagues, he felt that the same attitude of mind expressed by Dr. Shambaugh was experienced by all conservative laryngologists. He never approaches a patient, whose profession is singing, to do a radical operation on the throat, without mental reservation. And yet he has operated on many such, always with satisfactory results.

He does not know what Dr. Kenyon's conclusions will finally be, some years hence, but it seems to him that he must evolve a new operation, by which the tonsil mucous membrane shall be carefully peeled off, like the peeling of an orange, the tonsil shelled out, and the mucous membrane carefully sutured to the capsule and to the pillars, thereby saving the entire sinus, well filled with epithelium. But even then he would not have the tonsil, which he seems to indicate somewhat dogmatically is necessary for perfect function and perfect anatomic relations of the throat. The practical clinical experience now of many years of many observers of many cases does not substantiate the essayist's anatomic contentions.

Dr. Charles M. Robertson said that in the study of the tonsils we must necessarily recognize that there are two types of tonsils—diseased and nondiseased, if there is any such thing as the latter class. Dr. Kenyon, in his paper, which the speaker thought would do a lot of good, and which he was glad to hear, gave two thoughts. One was, as Dr. Corwin stated, that the tonsil must necessarily have a block behind it to support it, and the other, that the tonsil in its enucleation produces more or less cicatricial tissue, which destroys the function of one or both muscles. In children we find tonsils that have not been diseased particularly—there are no adhesions—and the tonsils should be removed, so that the cicatricial tissue would be at a minimum. In adults who have had recurrent attacks of tonsillitis, Dr. Kenyon must remember that these are not cases of normal tonsils. There may be a lot of cicatricial tissue there before the tonsil is enucleated, that has already interfered with the glossopalatal and glossopharyngeus muscles. We find cases even that have had necrosis of these tissues and necrosis of the superior constrictor muscle. There are more than forty cases on record in which the necrosis has been so great that there has been aneurism and rupture of the internal carotid artery and death. In a case like that you could not expect to operate and have normal tissue afterwards in the muscle. The muscle is already weak or scarred. Of course, if there is a large tonsil, you must expect a lot of contraction. After operation is completed, you have a raw space of perhaps three-fourths inch high by two inches around on either side of the throat. If that is going to heal by granulation, there is going to be contraction, of course, and that must necessarily pull the soft palate, and must necessarily affect the glossopalatal and glossopharyngeus muscles. But here is the point: In all those cases where there is contraction after operation, there is an aftermath in the months following, in which the cicatricial tissue softens, just as any scar tissue softens.

As regards operating on singers, Dr. Robertson does not understand why men should fear it. He would operate on a singer just as soon as on anybody else, or a little sooner, perhaps. A person who has a voice that has been trained with a chunk of tonsil in it is no singer, or will not continue one. If that person can sing today, in ten years from now he will not sing. That is universal. He would like to see the men in Chicago make a point of that. The men in the East are all hypnotized with the fear that they are going to lose people's voices. Dr. Robertson has operated on dozens of singers, and they were all improved.

In many cases there are cheesy masses in the tonsil, which means an infected gland, and you can have an inflamed gland without a manifestation of hypertrophy of the tonsil, infecting the pillar, and also the muscle, and what is to prevent the muscle from atrophying, because it is an inflamed muscle? We have that in other parts of the body, why not there? He just mentioned this to Dr. Kenyon, not that he was antagonistic to him, but he would like to have him bring these points out.

It has occurred to him that it would be a nice thing if someone would evolve a plan by which a little skin graft could be planted immediately after operation.

Dr. Robertson does not believe that the present tonsillectomy is the ideal operation. He believes, with Dr. Kenyon, that the tonsil surgery is in its infancy. However, he does not believe there is such a thing as a healthy tonsil after the age of eight or ten. We should have the tonsil all out; we should have a mucous membrane that is absolutely clean, with epidermis all around, and lining mucous membrane, so that the pillars would be free from each other. He dislikes to see the two pillars coalesced. Those are the cases that produce the voice injury, if there is any such thing. It is just as important to operate correctly on a talker as on a singer.

Dr. J. Holinger wished to refer to two points. The first concerned the injuries to the voice after tonsillectomy. Usually these people know of some person whose voice was injured. The speaker never had a chance to verify such a statement personally. The second, the formation of a scar depends entirely upon the individual. In some patients you see, with a minimum of traumatism, an enormous scar; in others, after extensive traumatism, very little or no scar. You see that after any operation in the cavities of the body or on the surface. But the scar depends also upon the case itself. If you have a deep supratonsillar fossa, you must expect a pretty large traumatism in order to close the supratonsillar fossa. If you do not close it, you will have a pouch afterwards, where all kinds of material can gather, causing trouble again. Therefore, if it is a question of trying to save the muscle by doing an incomplete operation, or the other way around, he could not say which was the more advisable.

Dr. J. R. Fletcher said that some of the members thought the operation had not been evolved which would save all of this membrane, and he wished to say that that operation has been evolved, namely, dissection.

He thought many of the members could show throats of patients they had operated in which the scar was very slight indeed, in which the action of the anterior and posterior pillars was absolutely perfect.

By careful dissection, the membrane is saved on the posterior pillar or palatopharyngeus muscle.

Regarding the anatomy of the palatopharyngeus muscle, it does not seem to be very widely recognized that there is a portion of the palatopharyngeus which acts as a levator of the tonsil itself. The speaker has dissected that out in autopsies and in nearly every case of tonsillectomy that he has done. That is the only muscle that is cut at all in doing a careful dissection. The palatopharyngeus muscle can be so stripped from the capsule that you can see the aponeurosis that covers it. There is a certain portion which has fibrous tissue lying between the muscle and the capsule of the tonsil.

As to the mucous membrane being deflected from the pharynx over the posterior portion of the palatopharyngeus muscle, then over the face of the tonsil, there is no such thing. The mucous membrane attaches itself to the posterior aspect of the tonsil, whether large, small or otherwise.

Regarding adhesions, the speaker does not think they are necessary, if a strict, rigid technic is followed. He believes the tonsillar fossa can be preserved, and the function of the muscles preserved also.

DR. KENYON, in closing the discussion, said that the function of the faucial muscles is probably dependent altogether more upon the capsule than upon the lymphatic tissue.

Permanent impairment of the speaking voice does occur from tonsillectomies. Some of the physicians in the room seemed to doubt it. Such impairment may turn out to be more frequent than we presume.

Again: It seemed to him that there was no reason why we should have an exclusively radical attitude toward the tonsil. What we need is a conservative attitude as well as a radical attitude, and it is for us to work out a conservative attitude for conservative cases.

The question as to the singing voice opened up such a large field that he would not then, at so late an hour, touch on the subject at all.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of April 18, 1916.

DR. OTIS H. MACLAY, THE PRESIDENT, IN THE CHAIR.

Case of High Degree of Retraction of the Posterior Half of the Drum Head as a Consequence of Old Adenoids.

DR. J. HOLINGER demonstrated a boy, M. H., sixteen years old, who complained of hard hearing, dizziness and noises in both ears for the last two years. Neither ear ever discharged. Large quantities of ear wax and epidermoidal scales were removed from both ears, especially from the right, where they were anchored in the region of the posterior upper quadrant of the drum membrane. Both membranes were considerably retracted. Hearing distance in the right ear was one meter for whisper; in the left, three meters. In catheterization the air entered with a soft sound in the right, with a harsh (normal) sound in the left ear, after which the aspect of the right drum membrane was as follows: The anterior half is normal, the posterior half shows the highest degree of retraction. The membrana flaccida shows the reflex of a cove. The handle of the hammer is in its normal position, but the posterior half of the membrane is tightly adherent to the inner wall of the middle ear. The short process is quite prominent, and a high posterior fold extends from it, sickle-shaped, to the posterior margin. Depressing the speculum downward and forward, one is able to look underneath this fold into a bag-shaped excavation of the membrane, extending into the aditus ad antrum. On the inner wall of this bag the long process of the incus, the stapes, and, extending horizontally backwards, the tendon of the stapedius muscle are clearly visible. The atrophic adherent part of the membrane is drawn tightly, like a glove over the hand, over all these parts. The left membrane is normal.

Considerable masses of adenoids and the tonsils were removed. Hearing improved in both ears—in the right to four meters, in the left to over seven meters for whisper. Rinne's test: a^1 right ear plus ten seconds, left ear plus twenty seconds. Weber-Schwabach: a^1 in the right ear positive; a in the right ear plus twenty seconds. The lower tone limit in the right is 26 v.d.; in the left ear, 16 v.d.

Explanation.—There is in the right ear one of the consequences of adenoids. Through absorption of air in the antrum and cells of the mastoid, the membrane became first retracted, then in the posterior half adherent, and extended bag-shaped into the aditus ad antrum, thus overlapping and later on replacing the normal lining by epidermis. Masses of epidermis gathered in the bag, and there is the beginning of cholesteatoma. The most forcible inflation through the catheter gives subjective relief, but does not change the picture. Had the speaker had a chance to remove the adenoids a year or so ago, normal conditions might have been obtained, as secured in the left ear. Now the boy is a chronic invalid.

DISCUSSION.

DR. GEORGE E. SHAMBAUGH asked Dr. Holinger whether he considered the condition of the drum membrane the result of reaction alone.

DR. HOLINGER replied in the affirmative.

DR. SHAMBAUGH said that the impression he got from the appearance of the drum membrane in this case was that of scar formation in the upper posterior quadrant. The most extreme form of retracted drum membranes which one encounters in the nonsuppurative types of middle ear troubles presents a condition where the handle of the hammer is entirely obscured, drawn up under the posterior fold. In Dr. Holinger's case the manubrium is still visible. The formation of a cholesteatoma in the pocket formed in the upper external margin of Dr. Holinger's case is the most interesting feature, and throws an additional light on the method of formation of this interesting clinical condition. It is apparently always due to the accumulation of desquamated epithelium and develops, as a rule, from epithelium which has invaded the middle ear cavities. Dr. Shambaugh has seen a large cholesteatomatous mass filling the inner third of the external meatus, where there has been no perforation into the middle ear.

Dr. Holinger's case resembles rather closely the cases of cholesteatoma developing in the attic, the origin of which has been cleared up by the work of Bezold. The membrana flaccida is very often sucked in against the neck of the hammer, to which it becomes adherent. It becomes very easily torn, and in this way forms a portal for entrance of epithelium into the attic. This eventually results in a cholesteatomatous formation.

Dr. Norval H. Pierce thought the case very interesting, the pathology of which has to do with the very keynote of the pathology of the middle ear. His understanding of the process of formation of cholesteatoma in this type of cases, as described by Bezold, is that the eustachian tube becomes closed. He asked Dr. Holinger if the tube was permeable in the case presented.

Dr. Holinger replied that it was.

Dr. Pierce asked if he could see the location of the adhesions.

Dr. Holinger replied that the anterior part of the membrane is covered, but not the posterior.

Dr. Pierce said that Politzer claims that in all these cases there is an absolute shutting off of ventilation from the part of the ear affected—not necessarily in the eustachian tube, but somewhere in the cavum tympani, so that no oxygen gets into that portion of the cavum; whether in the tube or whether due to adhesions somewhere among the various ligaments of the epitympanic space and the tubal ostium, makes very little difference. The fact remains that there is no ventilation or no ingress of oxygen to that particular part of the cavum which is affected. As the members all knew, the theory is that the oxygen is absorbed and carbonic acid given off by the mucous membrane in the cavum. It is well-known that carbonic acid is a great stimulus to muscle fiber, and probably in these cases this surplus carbonic acid contributes to the abnormal contraction of the tensor tympani, adding to the retraction of the tympanic membrane. There are two entirely dissimilar otoscopic pictures presented by these cases: One in which the manubrium mallei is in an approximately normal position as regards its lateral relations, the other in which the manubrium is greatly foreshortened, or scarcely visible, in advanced cases, it having been retracted inwards and upwards toward the eustachian tube orifice. The explanation is this: In the former cases the manubrium has been at the very beginning of the process bound down to the promontory by a plastic inflammation, rendering the lateral dislocation impossible. In the latter cases no such adhesion has occurred, so that the plus atmospheric pressure outside of the tympanic membrane and the contraction of the tensor tympani has been unrestrained. Cholesteatoma are more likely to form in the former class of cases. Atmospheric pressure remains the same in the external auditory canal—sixteen pounds to the square inch—and this pressure produces pressure atrophy. Pressure atrophy has its greatest effect on the least resistant portion of the membrane—Shrapnell's membrane. Shrapnell's membrane is composed of two layers, the skin and the mucous membrane, whereas the pars tensa is composed of three layers—the layer from the dermis of the external auditory canal, the middle or fibrous tissue, and the layer derived from the mucosa of the cavum. Therefore, the pressure atrophy has its greatest effect upon Shrapnell's membrane.

Before cholesteatoma can occur in these cases we must have a perforation, which perforation occurs in Shrapnell's membrane as an end result of pressure atrophy. After perforation occurs, then the epithelium from the external auditory canal and the tympanic membrane crosses through the perforation and into the aditus and epitympanic space. These are the cases of adhesion in these early stages that recently have attracted attention—in New York especially. It has been claimed that considerable benefit as to hearing is derived from mobilizing the handle of the malleus from the promontory. An incsiion is made below the long handle of the malleus, and with a ring encircling the malleus the adhesions are broken up. We all know that this is only a small part of the pathology of such a condition. Dr. Pierce's experience has led him to believe that it is impossible permanently to mobilize these cases, and in cases of threatened cholesteatomatous formation it is a question in his mind whether ossiculectomy is not advisable, to prevent the formation of cholesteatomatous masses up in the antrum. He has been able to remove the cholesteatoma in a couple of instances by injecting salt solution under the periosteum of the external auditory canal. There is usually a break in the periosteum that covers the outer surface of the epitympanic space, and this fluid apparently travels under the periosteum of the external auditory canal into the epitympanic space, and then washes out the cholesteatoma through the perforation. In one case, at least, this method was followed by an apparent cure. In other words, the cholesteatoma did not return for a considerable time afterwards.

These cases are very elementary, but very practical, and the speaker was glad that Dr. Holinger had brought the case before the society.

Dr. Holinger, in closing the discussion, said that the difference between the condition present in his case and that reported by Dr. Shambaugh is in the fibers of the membrane extending downward and forward from the end of the handle of the mallet. If these fibers are strong enough to withstand the traction of the tensor tympani muscle, the handle of the mallet will remain in place; if not, or if there is a perforation of the light cone, the handle will move upward and backward. In his case these fibers saved the anterior half of the drum head. The posterior half became retracted, atrophic, adherent and formed what Bezold called a "cholesteatoma of the external meatus."

Paper: Massage of the Eustachian Tube.*

By Alfred Lewy, M. D.

DISCUSSION.

Dr. J. Holinger thought a paper of so much importance as that presented by Dr. Lewy should not go without discussion, especially at the present time, when we actually see so many of these cases. The epidemic of influenza of last winter was combined in a great many cases with middle ear suppuration and affection of the eustachian tubes. Many of them belonged to the category spoken of by Dr. Lewy, with more or less retraction of the drum head, and very distressing subjective noises. The speaker did not know whether the other members saw as many of such cases as he had, but it seemed to him that sometimes for days he hardly saw anything else except secondary changes in the tube and middle ear, caused by chronic or subacute nasopharyngitis. He had not had the courage thus far to massage the tubes directly, although the indication was present. The question in his mind has always been, is it probable that we accomplish more in these cases by an active treatment than by waiting? Therefore, he welcomed the paper. It showed that with the careful technic described in the paper, Dr. Lewy did not see very much result, and the other members need not expect much more from their endeavors. The socalled stenosis of the eustachian tube has been discussed very extensively. The Schwartze school insisted on using the bougie in the eustachian tube. Bezold has shown that there is a curve in the eustachian tube. This curve is sometimes very sharp, so that a bougie may get caught, and not pass. This does not necessarily mean a stricture. The pathologic changes of the eustachian tube are not in the course of the tube, but at both ends, either at the tympanic end or at the nasopharyngeal end. The nasopharyngeal end can be reached with Dr. Lewy's method, often with good success. The work that has been done so far with the nasopharyngoscope is very promising of further success.

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*See page 898.

DR. JOSEPH C. BECK said that a few years ago, when he began to examine the eustachian tube by the present nasopharyngoscope, then known as the Valentine salpingoscope, he saw a recess behind the posterior lip of the eustachian tube that was never visible to him before. At that time Dr. Pynchon told him that he had been practicing what he called massage of the eustachian tube and the lymphoid tissue which he found in that region. But for the last three years, since the speaker pulled forward a palate in the adenoid operation, he has frequently seen the socalled Rosenmüller fossa and the posterior lip, a condition that was never clear to him by any mirror examination.

After acute or chronic inflammation of the nasopharynx there is an infiltration in the muscles of the tube, so that the normal action does not take place, and then by this massage action, sweeping around these three different strokes, normal action may be brought about. Too little attention is paid to the eustachian tube, particularly to the muscular apparatus of the tube.

He thinks that the result oftentimes obtained by the removal of the tonsils is due to the change produced on that little muscle, which comes from the palate, that goes around the eustachian tube opening. Hence the parts have again reestablished themselves.

Massage in these chronic cases improves the condition temporarily, but when left alone the condition returns. The result is on the muscle apparatus more than anything else.

DR. H. KAHN said the question of massage of the eustachian tube is not new. The method described by the essayist is novel. Ten or twelve years ago Urbantschitsch massaged the eustachian tube by passing a eustachian catheter; through this a celluloid bougie was inserted, and then with a backward and forward movement of the bougie five, ten or fifteen times, as desired, hyperemia was established, and adhesions were also broken up. Urbantschitsch also practiced a backward and forward massage of the nose, with a mechanical vibrator, which reached back around the end of the tube through the length of the lower meatus of the nose. This vibrator was armed with cotton and an oleaginous substance, and rubbed the lower end of the turbinate. Dr. Kahn could not see that the method does anything more than establish a hyperemia, or to make a postnasal application in a definite way.

DR. ROBERT SONNENSCHEIN, in closing the discussion for Dr. Lewy, said he hardly felt in a position to discuss the paper. He knew that some men have practiced massage of the eustachian tube on the same principle that pneumomassage is applied to the external ear—by passing a catheter into the eustachian tube, and then applying the tip of the apparatus which transmits the pneumomassage to the end of the catheter, and massages in that manner.

Whether the efficacy of all these methods is due to the fact that a hyperemia is induced, or whether the exercise of the muscles, as suggested, is the prime factor, he did not know, but thought that perhaps both had something to do with it.

The fact mentioned by Dr. Beck regarding temporary improvement of hearing after removal of tonsils and adenoids is probably seen by many. Nothing has shocked the speaker more than to see a wonderful improvement of hearing after removal of the adenoids, and then, a year later, retrogression again. Whether this was due to the fact that a small amount of lymphoid tissue is left in the fossa of Rosenmüller, or to the fact that the muscles have renewed their former tone or lack of tone, he could not say.

Paper: Difficulties and Complications of Sphenopalatine Ganglion Injection.*

By Harry L. Pollock, M. D.

DISCUSSION.

Dr. Robert Sonnenschein said that, while he has never injected the sphenopalatine ganglion, he wanted to report a very interesting and absolutely typical case of the syndrome described by Sluder. In this case there evidently was a dehiscence of bone covering the ganglion, because the ganglion responded to treatment in a way that showed that it must have been exposed. The patient, thirty years of age, a married woman, came to the speaker about three years ago, and, without suggestion or direct questioning on his part, gave a history of the typical symptoms of the syndrome of Sluder. There were most terrific neuralgic pains, starting in the region of the cheeks, going into the mastoid, down the occiput, shoulder and into the arm. In order to relieve this, various dentists had removed all of the upper teeth, without benefit. Twice a week she had such severe paroxysms that she was obliged to stay in bed, and any exertion, such as going down town, invariably brought on a paroxysm. Having heard Sluder about a year or two before that describe the condition, the speaker at once connected the two, and on going up beneath the middle turbinate bone with the posterior end of a small applicator and touching this region, a most frightful paroxysm was brought on. He then used a saturated cocain solution, as suggested by Sluder, and in a few minutes relief was obtained. He then used formalin two or three times a week for six or seven applications, beginning with one-tenth of one per cent solution. The paroxysms diminished and then disappeared, so that for two years she was practically free from pain, except for very slight occasional pains which did not incapacitate her. Two or three months ago she returned, with the statement that she had one or two paroxysms, which, however, were nothing compared with the original ones. Dr. Sonnenschein then applied the formalin again for a few treatments, and the pains disappeared.

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*See page 958.

This case had the typical symptoms and yet yielded to these simple topical applications. There was no hysteria present in the case, but a sad reality in the removal of all of the upper teeth in a young woman of thirty, for a condition which was not benefited by their removal.

Dr. H. Kahn said the whole question under discussion was of much interest to him, particularly the mechanism of cocain when applied in this region—the posterior end of the middle turbinate—and about the region of this ganglion. What is the mechanism? Why does the cocain act? There must be some intimate connection of the outside with the inside.

Dr. Beck said that they had obtained results in other cases, but those were the best where the cocain was used.

Dr. Kahn said he would like to know the mode of action of the drug.

Dr. Norval H. Pierce said he had had very little experience in this matter. He had had some, however, and had seen Sluder work. One of Sluder's cases he remembered, of a physician who had to give up his work for a long time on account of this pain running down into his right arm. This man told Dr. Pierce himself that he was cured, the cure coming on after the second or third injection of his sphenopalatine ganglion. He also saw a case of nervous affection of the eyelids injected by Sluder, and Sluder afterwards wrote him that the case had been entirely cured up after a week.

The speaker had had one case that he rather hesitated to say anything about, because he was quite sure he did not get into the sphenopalatine ganglion. This patient, a man, was a highly nervous and imaginative individual. He suffered from ethmoid disease and the arm pain, which was not neurotic in character, however. He would describe it by saying that a numbness would start in his face, and then a sensation like the buzz of an electric bell would seize him over the collarbone; this would be followed by numbness and sometimes pronounced pain running down the arm. Dr. Pierce told this man that he had recently seen cases of that sort treated, and that if he would consent to the experiment, he would be glad to try this method of treatment. The man readily agreed to it, and Dr. Pierce tried to get into the sphenopalatine ganglion. The patient had a very open nose, and he could see his way very distinctly. He put all the pressure that he thought was justified on a very heavy needle and tried to get in. He was quite sure, however, that he did not get beyond the periosteum. However, a week afterwards the man came back and said that he was greatly improved. The telephone bell effect had disappeared, and all he had was a slight constriction around the left forearm, and he insisted on another injection, which Dr. Pierce did, in the same manner, in the same dense bone. In another week the man reported that he was practically all right, and was greatly pleased. Dr. Pierce did not feel that he could ascribe the effect to the injection of the ganglion.

One thing he wished to speak of, however, was that there was quite a copious initial hemorrhage.

All these cases of vague pain in the head, and strange abnormalities of vision are almost always connected with instability of the nervous system, which, on this account, are probably often benefited by any extraordinary method of operating. We should go very carefully about this whole matter before drawing fixed conclusions.

Dr. Louis Ostrom, Rock Island, said he had seen Sluder work, as well as a few others, and had seen what he considers a neglect of detail of technic that is not permissible in other work, namely, leaving air in the needle. Air is left in the needle to the amount equaling seven drops of water. Unless this air is expressed and the needle filled with the solution to be used, you are injecting air first. He has seen trouble following the use of sphenopalatine injections, and he felt it was due entirely to this neglect. His method is to fill the needle with the solution to be used and then cork one end. The needle is then introduced, syringe (already filled) attached, and he knows how much he is injecting. He thinks that is just as important in this connection as in giving an ordinary hypodermic, if not more so. It is all a matter of technic.

Sluder has his patient in the upright position. It is not an easy matter to make an injection, with a movable head, and know just exactly the force that you are applying. If you have the patient in the recumbent position, then make the injection, you have absolute physical control of the situation.

Dr. Ostrom believes these two points are just as important in the technic as the location. When you say the posterior tip, he believes that that also should be restricted as to what is meant by the posterior end of the middle turbinate, because you may have a very large tip of the posterior end, and unless you keep the exact location of the sphenopalatine ganglion in mind, you may go wrong.

DR. JOSEPH C. BECK thought the most important part in the cases that were reported is the effect of the alcohol on nerve tissue and on the neighboring nerves. The sphenoidal fissure and the pterygomaxillary fossa are in close proximity, and so is the sphenoid sinus, to a number of nerves, particularly the optic nerve. He believes these injections are made very frequently without any regard to vision. That is, the vision is not taken before or after these cases are injected, and the question is to be answered as to what effect alcohol has on the various nerves. Dr. Norcross presented a paper to the society on the injection of the superior laryngeal nerve, in which he reported hypoglossal paralyses or paralyses of the tongue, and Dr. Beck has had such an experience, and we have heard about the abducens paralysis. In this injection of sphenopalatine ganglion the optic nerve gets a bath of alcohol—at least, the adventitia of the optic nerve does—and it is yet to be proven what the effect of the alcohol is on the optic nerve. We know the hypoglossal and abducens could be affected. He wished to report a case of facial paralysis following the use of trichloracetic acid. He completed a radical mastoid operation, in which he had previously closed the pharyngeal end of the tube, and got a complete closure of the tympanic membrane in a case of continuous discharge from the ear. The perforation healed up, but soon after that there were recurrences of fluid accumulation in the ear, with typical attacks. Rupture ensued; then the patient was well for several months. Then there was a good deal of pain in the mastoid. The speaker opened up and did a radical mastoid. In order to end the trouble he used Yankauer's curette, and then with a Holmes' applicator very carefully applied the trichloracetic acid into the tube to further insure obliteration, but undoubtedly must have spilled some of the trichloracetic acid in the region of the facial canal, or in some dehiscent portion of it. At any rate, the patient had a transient facial paralysis, which took only a short time to recover. The facial nerve might have been injured during the mastoid operation, but he doubted it. He took it to be a case of facial paralysis from trichloracetic acid, analogous to the action of alcohol.

Dr. Beck could say frankly that the results from these injections are uniformly poor. They are not lasting. Many of them are failures, especially those that are done for vague pains in the head. Quite a few have been benefited, but striking results, such as he has heard reported by others, he has not seen. The condition is apt to recur, because you do not get a destruction, as in the use of osmic acid. There is only a temporary destruction of the neurons, or part of them, which is followed by a reformation of the structures.

The question of hemorrhage is, of course, important. We must be careful not to injure that branch of the posterior pharyngeal that goes down in close proximity to the sphenopalatine ganglion. In one case the speaker injected the ganglion in a patient, and a week later the alarming hemorrhage occurred. She had had some bleeding at the time of operation, but this hemorrhage must have been due to secondary infection.

Dr. J. Holinger had made several of these injections, and could only coincide with the other speakers. The results were not as he had expected. He especially recalled one patient in whom he injected the sphenopalatine ganglion three times, at intervals of five weeks. The symptoms were typical. He was sure that he followed the technic to the very letter. There was no benefit derived by the patient. He had had the same experience with the two other cases he had injected.

Dr. Pollock, in closing the discussion, was glad to hear so much adverse criticism, but wished to call attention to a paper, previously read by him, in which he had stated that the results were not as brilliant in his hands as those reported by Sluder, and that was his reason for reporting his failures, so to speak. However, he has had some good results. Uniformly, the results have not been so good as he would like them to be, but he could not quite agree with most of the men, because he has in a great number of cases obtained good results.

In regard to Dr. Kahn's question as to why the application of cocain in this region results in temporary cure of the patient, or relief of symptoms, he wished to say that the cocain has a very strong penetrating effect on mucous membrane, and the sphenopalatine foramen is right in that neighborhood. He tries to put the cocain up against the sphenopalatine foramen. There is an opening in the bone in the posterior wall, and there is nothing but a little mucous membrane to penetrate through.

Dr. Kahn asked if Dr. Pollock got the same result in thick bone.

Dr. Pollock replied that there is a sphenopalatine opening in the foramen in that region, where the nerve comes out—the nerve that supplies the mucous membrane of the turbinals; the cocain is applied in that region, and it penetrates.

Sluder has also advocated applying a solution of salicylic acid and various other medicaments that penetrate and have effect on these nerves. That is the reason we get these results. In all those cases where instantaneous relief is experienced, the results have been good.

Dr. Pollock did not wish to be understood as saying that when a patient comes with these pains, the sphenopalatine ganglion is injected immediately. That was not the idea of the paper.

He referred to a case he saw in consultation with a doctor from St. Louis, which was one of typical sphenopalatine neuralgic pain. This doctor had diagnosed the case, and had had an X-ray picture taken, which showed that the third molar was impacted, in a young lady, about twenty-three years of age. He advised removal of the tooth, but she objected to it. They brought her to Chicago, and Dr. Pollock saw the young lady, and he also advised removal of the tooth. This was done, and immediately all the symptoms disappeared. It is the same way when there is a suppurative ethmoiditis or sphenoiditis causing irritation of this ganglion. All points of irritation are removed first, and if nothing results, then resort to the sphenopalatine ganglion injection. It is a sort of a measure of last resort.

With regard to Dr. Ostrom's remarks about the head being movable, the speaker always has a nurse to support the head. In very difficult cases he gets a hammerlock on the patient. A great deal of force has to be used in most of these cases, because the bone is sometimes so thick that even with a hammer you cannot drive the long needle into the bone. Dr. Sluder reported one case in which he actually drilled through into the pterygomaxillary fossa, and curetted out what he took to be the sphenopalatine ganglion This is the only case of drilling on record in this operation.

Dr. Pollock does not think there is any danger of using too much pressure, because you can feel that by your pressure sense. It is just the same feeling as in penetrating an antrum. Of course, the bone is thicker in most cases than the antral wall, but you can feel the needle slip into place.

In regard to Dr. Beck's remarks about the effect of alcohol injection upon the different nerves, the sensory and motor as well as the optic, the speaker had had no experience in this line. Otto May of London reported his results in the *British Medical Journal.* He injected alcohol in from fifty to ninety per cent strength, using from one-half to three-fourths centimeters, and injected into the supraorbital, Gasserian ganglion, the sciatic and anterior crural nerves in cats and dogs. He experimented on from seventy-five to one hundred animals, but arrived at the conclusion that alcohol injected into the trunk of a peripheral nerve produces a more or less local necrosis of the nerve at the point of injection. The degeneration that occurs is not an ascending one anywhere. The nerve above the point of injection remains normal. The cells at the origin of the fibers may show some degree of chromatolysis, but do not exhibit signs of permanent injury. The condition produced by such injection is more favorable to regeneration than that resulting from simple section. That is, by the injection you get earlier regeneration than from injection without suturing the nerve again. The rapid regeneration of the nerve is retarded somewhat by the fibrosis which occurs, to a greater or less extent, in every case of alcohol injection.

About the Gasserian: It is apparently impossible by a single injection of alcohol to produce complete necrosis of the Gasserian ganglion, its dense texture preventing complete infiltration. Alcohol tends to find its way underneath the sheath of the ganglion to the proximal root, which is affected to a greater degree than the actual Gasserian ganglion cells. There is a regeneration that occurs even after injection of the sphenopalatine ganglion. That is why some cases that are injected seem to have good results, and after a period varying from six months to two years they return with the same symptoms to a mild degree. They are reinjected, if necessary, two or three times. One patient the speaker has injected five times. Once you strike the ganglion, there is no use injecting further. The ganglion is a very small particle of the nerve tissue, situated in quite a large fossa. It is not very often that the speaker has struck it the first time. If you inject the alcohol and it permeates around, it does not affect the ganglion the same as if the needle went directly into the ganglion, which is demonstrated by the excruciating pain. When the ganglion is struck, uniformly good results are obtained, but the results are not as good as expected, nor as good as Dr. Sluder claims. However, Dr. Pollock said that there have been a large number of cases where relief from the pain was experienced.. Of course, all cases of hyperesthetic rhinitis got relief, and a large percentage of the cases of neuralgia were benefited.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of May 22, 1916.

DR. OTIS H. MACLAY, THE PRESIDENT, IN THE CHAIR.

Case of Increasing Loss of Hearing—Left-Sided Facial Paralysis—Hoarseness—Tumor in Right Parotid Region—Hemiatrophy and Paresis of Left Side of Tongue.

DR. NORVAL H. PIERCE demonstrated the case of a man, aged sixty-one years, who consulted him because he was getting hard of hearing on the left side. Tuning fork tests: Left—Weber-Schwabach, twenty-five seconds, lateralized to the left ear; Rinne, ten seconds osseous conduction, and twenty-seven seconds aerial conduction. Right—Fifteen seconds osseous conduction, and forty-five seconds aerial conduction. Low tone limit on both sides, G^2 (twenty-four vibrations a second). High tone limit on both sides decreased. The high tones on the left more decreased than the right. Whisper heard on left side at four to twelve inches, including high and low pitched words. Right, ten inches to three feet. The same type of disease is present, therefore, in both ears, a type that has been named by Bezold as dysacusis—both the middle and internal ears being involved. The speaker found a facial paralysis on the left side, and a tumor in the right parotid region, which tumor was immovable from the deeper structures. Only the skin was movable. The tumor was hard and somewhat nodular. The tongue showed a hemiatrophy on the left side, and in projection was directed to the left side. The man complained of hoarseness, and on examination it was found that he had adductor paralysis of the left vocal cord.

Dr. Pierce's analysis of the case was as follows: The tumor is in all probability the socalled mixed tumor of the parotid, which is a very curious kind of tumor, affecting the parotid and the submaxillary glands, most frequently the parotid—about seventy-one per cent parotid, and the rest submaxillary.

In the early stages such tumors are movable within the parotid tissues. It is hard or soft, according to its composition; is somewhat nodular and covered with well-pronounced limiting membrane or capsule, from which capsule strands of connective tissue are thrown into the tumor tissue, forming lobules. The microscopic character of the tumor has been the source of a good deal of controversy, some claiming that it is derived from the connective tissue elements, others that it is derived from the endothelium or epithelium elements. Inasmuch as these tumors contain fat, cartilage and sometimes bone, the speaker believes that the school represented by Volkmann, which holds that they are of embryologic origin, is correct. The peculiar histology is this, that the tumor proper is composed of cylinders formed of epithelial or endothelial cells, columnar in character. The embryologic displacement theory is substantiated in this particular case by the fact that some time ago the man had a tumor removed from the neck, which the surgeon told him contained hair—in other words, a dermoid, which is derived from an embryonal matrix.

The facial paralysis is due to involvement of the facial nerve on that side by the tumor. The hypoglossal involvement is due also to involvement by the tumor, as the nerve passes down the retromaxillary fossa. The tumor has nothing whatever to do with the nerve of hearing or the hearing apparatus, other than that it is encroaching upon the external auditory canal and closing it up. It is scarcely possible to see the tympanic membrane on that side. This opinion is further strengthened by the fact that the same type of disease is present on both sides.

The facial paralysis is not due to anything going on within the temporal bone proper, but is due to a peripheral involvement of the tumor, which is proven by the fact that the palate is not involved. The glossopharyngeus is not involved, but the hypoglossus is involved, which is due to invasion by the tumor of the retromaxillary fossa.

The disease of the larynx is an entirely different affair, and is in all probability due to some trouble within the thorax, although a somewhat superficial examination did not disclose anything wrong in the way of a large aneurism.

The Wassermann in this case is negative. The man disclaims any venereal disease.

In the speaker's opinion, the treatment of the tumor is of little value. In all probability, it is undergoing a malignant change, as such tumors are very liable to do. When such change takes place, there is very little to be done, in a surgical way. Possibly X-rays could be tried. The type of hardness of hearing present in this case is uninfluenced by treatment.

Case of Previously Existing Ethmoid Labyrinth Suppuration With Polypoid Tissue, With Probable Invasion of the Orbit by a Malignant Process.

DR. NORVAL H. PIERCE: This patient is fifty-six years of age. Five and one-half months ago he was held up and struck on the head. He has a discharge from the left side of the nose, with symptoms of nasal stoppage. After the trauma a neuralgia developed in the upper and lower branches of the fifth nerve, which was especially marked over the frontal division of the nerve. Ten weeks after the injury he had an operation on the supraorbital nerve, when it was dissected out from the supraorbital foramen. For a time following this operation he was better. Then the pain recurred, and at the present time is as bad as ever. Gradually double vision developed, so that at the present time he sees double when the object is downward and to the left. In examining his eyes and making them go through their excursions, it appears that the inferior oblique is involved. The eyeball is slightly raised above its fellow. There is an enlargement over the left cheek (malar bone).

Examination of the nose reveals polypi, which have been removed several times. Their removal was accompanied by a very profuse hemorrhage.

The question is, what has occurred to this man? He evidently had the ethmoid disease long before the accident. X-ray examination shows that there is a shadow in the region corresponding to the left frontal sinus, left maxillary sinus and left sphenoid. Is it a case of malignant growth beginning after the injury to the skull? Is it an inflammation of the structures of the orbit from the previously existing ethmoid disease, or is it a hemorrhage at the base, which has come on after the injury or at the time of the injury, and is now just becoming organized and causing pressure on the nerves at the base?

Examination of the tissue taken from the nose showed nothing malignant.

Dr. Pierce's diagnosis is this: Previously existing ethmoid labyrinth suppuration, with polypoid tissue, and a very strong likelihood that there is an inflammatory or malignant process now invading the orbit. It is not possible to differentiate very well by the nerves involved, inasmuch as the superior branch of the third nerve accompanies the ophthalmic branch of the fifth. If it were the superior oblique, we could say that an inflammatory condition in this region could very easily involve both of these nerves, but if he is right in believing that it is the inferior rectus, then that nerve is situated at some distance from the ophthalmic division of the fifth. It would require quite a good deal of area to be covered by any process whatever to involve both of them, and there would be vastly greater local evidence of involvement of the orbital structures. This would suggest, to his mind, a malignant rather than an inflammatory trouble.

Glaucoma might be thought of also. The tension of both eyes is about alike, however. The eye grounds are normal.

This case should be very carefully weighed before attempting to do anything in the way of an operative procedure. However, Dr. Pierce is of the opinion that whatever is done should be done externally.

DISCUSSION.

Dr. H. Kahn said that the second patient had come to see him about two months ago, having just previously consulted Dr. S. Kuh, who could find nothing neurologic except the involvement of the fifth nerve. Dr. Kahn sent the man to the hospital, where a large number of very large polypi were removed. After the first sitting the hemorrhage was quite profuse, enough so to be very alarming. He was kept at the hospital for a day or so, and the symptoms apparently improved—the headache got better, and he could read a little bit. He did not have double vision at this time, and did not tire so much on reading. A day or so after the first operation, Dr. Kahn took off a piece of the middle turbinate anteriorly and opened up the ethmoid, which was immediately followed by a great gush of pus. Following this there was quite a disconcerting hemorrhage. Then the man was let rest again. From day to day, in the course of a week, the speaker tried to clean up the ethmoid. When the man left the hospital the pain and double vision had disappeared and he was fairly comfortable. During this time Dr. Snydacker examined the eye, and Dr. Kahn was of the opinion that a diagnosis was made of superior rectus involvement and beginning glaucoma in both eyes, with a cupping of the discs. When Dr. Kahn had the man under his care there was not such a pronounced proptosis as at present. His pain was relieved by a little aspirin, or some mild coal tar derivative, and he was kept fairly comfortable in this way. Of one thing Dr. Kahn was pretty sure, namely, that there was no malignancy reported. X-ray taken at that time showed substantially the same as that taken by Dr. Pierce. Puncture of the antrum was negative, and the frontal sinus findings were also negative on washing out. So far as could be made out, the whole thing most likely lay within the ethmoid. What the exact condition is, he could not say.

Dr. Robert Sonnenschein said that at the request of Dr. Kahn he had had the privilege of seeing the second patient, and also saw the X-rays. He had simply examined the man's nose, without cocaining him, and felt that it was a question for further investigation. But, as Dr. Pierce had said, if anything were done in the way of radical procedure, an external operation would probably be the thing desired.

Dr. J. Holinger, regarding the first case demonstrated, asked as to the condition of Steno's duct. He wondered if there was any abnormal discharge or composition of the saliva.

Dr. Pierce said the discharge was no more than normal. It had not been examined chemically.

Dr. Holinger, regarding the second patient, asked as to the condition of the nasopharynx. He thought it would be a favorable case in which to make the examination of the postnasal space demonstrated in Champaign. If Dr. Kahn could do so, he would like to have him tell the society whether there had been any progress of the condition.

Dr. H. Kahn said there seemed to be more enlargement; at any rate, it seemed to him that there was more proptosis than when he had seen the man before.

DR. MACLAY asked as to the condition of the nose at the present time—whether there was any return of the former condition.

DR. KAHN replied that he had not examined the man for some time.

DR. PIERCE said there was a return of the ordinary mucous polypi.

DR. PIERCE, in closing the discussion, said that all knew that a sarcoma or carcinoma of the ethmoid cells may be accompanied by the ordinary myxomatous growths that are indistinguishable microscopically and macroscopically from the growths that accompany the ordinary purulent inflammation of the ethmoid labyrinth.

(NOTE.—August, 1916, on operation, carcinoma involving the ethmoid and orbit was proven microscopically.)

Papilloma of the Right Vocal Cord.

DR. ROBERT H. GOOD showed a patient, a young girl, aged seventeen years, who had had a papilloma on the right vocal cord, which remained after several operations. The cord was thickened, especially the ventricle band, and the cord itself was reddened. When the patient phonated there was but slight movement of the cord. The patient had been in the Cook County Hospital since last September, and she had a number of operations for removal of the papilloma while there. She came to Dr. Good three months after the last operation, which was performed by Dr. Beck, and had not been able to speak above a whisper for a year. The speaker did not expect to be able to do anything, but used fulguration and sesquichlorid of iron, ten per cent. He first fulgurated for ten minutes, combined with the sesquichlorid of iron, and to his surprise one week later she was able to talk some. He has kept this up, and now she can sing.

Dr. Good learned of sesquichlorid of iron being used in this way in Edinburgh in Turner's clinic, so he thought he would try it in the case demonstrated, and certainly it has done wonders. The little papilloma has gone, and the right vocal cord moves well now.

Case of Double Dacryocystitis.

Dr. Good's second patient was a young woman, who came to him suffering with a double dacryocystitis. Only one side had been operated by Dr. Good. He did the Yankauer operation, removing the anterior end of the inferior turbinate. First he put in a grooved probe through the lacrimal duct, which he used as a guide, removing the inferior turbinate about a quarter inch further back than the probe. Then he chiseled the entire internal bony wall of the ostium away, and then the entire bony wall over the sac. After the bone was all removed, he took a knife and ran along the groove in the lacrimal probe. Since this operation the patient has not had the least trouble.

Dr. Good's reason for not doing the West operation is because Yankauer has brought out the point that frequently it is the bony wall around the duct, down below, that makes pressure upon it, and he says if this bony wall is removed, so that there is no more constriction at the bottom of the duct, the natural opening is reestablished. He had to say, however, that as yet if he injects argyrol through the cannula it comes out through the middle meatus. But the patient has not had the sign of epiphora on the operated side. The other side is still suppurating, as before.

Before coming to the speaker the patient had had the ducts dilated twenty-eight times, so that there was absolutely no use for further attempt along that line.

Dr. Good operated last November. He expects to operate the other side as soon as the patient wants it.

Specimen of a Little Sieve Removed From the Trachea.

Dr. Good then showed a specimen of a little sieve which he had removed from a patient's trachea three days previously. The sieve had been in the trachea for three weeks, almost without symptoms until the last week. The peculiar thing about the case was the character of the symptoms produced by this foreign body. The child came to his office, carried by the father. She was ten years old. Pulse, 150; temperature, 99.5°, and gasping for breath. He could not understand how such a small object could produce so much obstruction in the trachea, unless it simply produced spasms. After removal of the sieve the child was entirely relieved.

DISCUSSION.

DR. OTTO J. STEIN asked Dr. Good how many applications of fulguration he had made in the first case, and also something about the technic. Fulguration has been used a great deal in these cases and discarded by many operators as of no value. He had tried it quite a few times. He did not believe he knew how to use it, as he never got any results of any value. He had seen by the literature that Yankauer is advising using it in a perfectly dry field. That seems to Dr. Stein to be the most logical way. It is used mainly under suspension to get the best results. Most of Dr. Stein's work was done under the indirect method. So he would like to have Dr. Good give some details as to how often used and the method employed.

DR. C. H. LONG has had some experience with fulguration, but his trouble has been that he could not continue it long enough at any one time. He could not continue it for fifteen minutes, even under cocain anesthesia, and the results were negative.

With regard to the Yankauer operation for dacryocystitis, he would like to ask Dr. Good how this operation differs from the West operation. West spent a good deal of time in developing a sac operation, which has quite a considerable technic connected with it. The speaker has done it on a dead subject several times, and his impression was that any opening near the anterior end of the middle turbinate that was made large enough would give an excellent drainage to the sac and cure the patient, as the opening most always closes. He did not see why it was necessary to have such an elaborate technic as Dr. West explained. The West operation was brought out principally for those cases that were not cured by extirpation of the sac, and drainage through the nose looks very simple. The principal objection to this operation is that the nasal opening closes and the patients are liable to a reinfection.

DR. ROBERT SONNENSCHEIN had had occasion to see the first patient at the County Hospital, but did not operate her. At the present time there is a decided change in the larynx as compared with what it was at that time. He had seen Drs. Beck and Friedberg attempt to remove the papilloma by the suspension method, but that day the patient did not tolerate the method very well, despite cocainization, and nothing was accomplished. However, a few days later she was operated, but Dr. Sonnenschein was not present. Just how much of the growth was removed at that time he did not know, but when he saw her the right cord was enormously thickened. It was not a papilloma with a pedicle, but a sessile, very broad-based tumor. At that time the girl was aphonic, and if fulguration did it, it was certainly wonderful. However, the speaker did not know how much of the growth was removed at the second suspension operation.

DR. OTIS H. MACLAY said, with reference to the West and Yankauer operations, that he had done the West a couple of times. While either one may bring about a result, it seems to him that the one that produces the least traumatism to the nose should be chosen, making a small opening rather than a long incision. The cases he had operated apparently gave good results. They were both pus sacs.

DR. GOOD, in closing the discussion, said he thought he had given seven treatments of fulguration, about ten minutes for each treatment. He was sorry he had used both the sesquichlorid of iron and the fulguration, because it left him in the dark as to which did the business.

As to the technic of fulguration, it is a very ordinary affair. There is a little platinum wire, or any wire, with a rubber cuff over the wire, which comes within a fourth of an inch of the tip of the wire. This is fastened onto a handle. The tube is bent and put into a handle, which goes down into the larynx. The patient holds the electrode in the hand. The fulguration handle has no connection at all. In the method Dr. Good uses you draw the electricity out of the patient, and therefore it is not necessary to have the field all dry, as in the Yankauer method.

DR. STEIN asked if Dr. Good has tried the method in any other cases.

DR. GOOD replied that this was the first case.

DR. H. KAHN asked if he noticed any reaction following the treatment.

DR. GOOD replied that he did not notice much reaction—perhaps a little less hyperemia.

Regarding the West operation, the speaker did not agree that all West operations mean cures. The patient is not suffering from the loss of the anterior end of the turbinate. Personally, he would rather have the two combined done on him to make more certain of good results.

Specimen of Carcinoma of the Pharynx and Esophagus, With Secondary Involvement of the Cervical Lymph Nodes.

Dr. G. M. McBean presented this specimen, supplementary to the remarks he had made at a meeting a couple of months ago, in the discussion of Dr. Norcross' paper on carcinoma of the pharynx. These remarks concerned a woman, forty-three years of age, whose condition had been diagnosed as tuberculosis of the larynx, pharyngitis, rheumatism, and almost everything else but the right thing. She had been having tuberculin treatments and treatment with vaccines; also large doses of aspirin. She had had a ventrofixation of the uterus done for pains in the throat, and a hysterectomy also. She had been urged to have her tonsils removed. The speaker could not remember all the things that were done. Since he had referred to the case two months before, the patient had died, and he had brought the specimen for the members to see.

The condition presented was a very rare one, being a carcinoma of the upper third of the esophagus. Carcinomas of the esophagus are usually in the lower end. It is also a rare condition in a woman of forty-three. The arytenoids were very markedly involved, but when the speaker had first seen the patient in March they were not involved. The vocal cords were at no time involved in any way, but the last few times the speaker saw her it was impossible to see into the larynx because of the immobility of the cartilage.

The postmortem examination was as follows: Only the organs of the oral cavity, neck and lungs were examined. Tongue apparently normal. Soft and hard palates and uvula normal, only somewhat paler than normal. Thyroid slightly enlarged, very pale, but free of macroscopic alterations. After opening the trachea anteriorly in the median line, there are several protuberances visible in the posterior wall. They form irregular nodular growths, which are still covered by the mucous membrane. They greatly diminish the lumen of the pharynx and of the upper one-third of the trachea. Through these nodular growths the vocal cords are partly obliterated and drawn to each other. There is no ulceration visible in larynx and trachea. The glottis and epiglottis are unaltered. After opening the pharynx and esophagus posteriorly in the median line, a large ulcerated neoplasm becomes visible. It encircles the entire esophageal tube, its upper border corresponding with the lower end of the epiglottis and extending downward for thirteen centimeters. The ulceration is surrounded irregularly by thick, indurated border. The surface of the ulcer is covered with foul-smelling necrotic, slightly hemorrhagic masses. The ulcer presents several nodular protuberances, varying in size up to a large cherry. These nodular masses are soft, of medullary consistence, obstructing entirely the tube. Below this neoplasm the mucous membrane of the esophagus is very pale. There is a chain of enlarged and somewhat indurated cervical lymph nodes visible on both sides, the nodes varying in size up to a lima bean. There is on each side one old string-like fibrous adhesion between both pleuræ, on the right side on the lower border of the middle lobe, and on the left side on the lower border of the upper lobe. Both lungs are larger and heavier than normal; especially the lower lobes are highly congested. The bronchi contain large quantities of a serofibrinous exudate, and in each lower lobe, centrally located, there is one rounded area, the size of a pigeon's egg, of reddish gray consolidation. The mediastinal and bronchial lymph nodes are unchanged. There is nowhere in the larynx, pharynx, trachea, esophagus, lymph nodes, or lungs the slightest evidence of fresh or old, or even healed, tuberculosis. The diagnosis made from postmortem examination was: Carcinoma of the pharynx and esophagus; secondary carcinoma of the cervical lymph nodes; old fibrous pleural adhesions; bilateral localized inhalation bronchopneumonia.

Paper: Remarks About the Development of the Diagnosis and Treatment of Sinus Thrombosis.*

BY J. HOLINGER, M. D.

DISCUSSION.

DR. NORVAL H. PIERCE did not think such a radical view of the matter should pass undiscussed. It seemed to him to mark a distinctly retrograde step in our ideas regarding the treatment of sinus thrombosis. It is against the general law of surgery, namely, where you find pus you should drain. There is no doubt but what a great many more sinus thromboses recover without operation than we have any idea of, but once there is a septic thrombus detected within the sinus, whether it is parietal or a floating thrombus, or whether it is an obturative thrombus, his opinion is that it should be removed, the same as a septic thrombus in a large vein or in any other accessible part of the body. If we do not do this, if we simply lay bare the sinus, and the patient recovers, he does not recover from our operative skill, but by the grace of good fortune.

The method described by Dr. Holinger has been tried, but not extensively, so far as Dr. Pierce knew. Its use has been limited to a very few men. It does not represent the modern idea of the subject by any means. These investigations on animals count for very little, Dr. Pierce thinks, as they are produced on healthy animals, and these thrombi in people, especially in the chronic forms, occur in people more or less reduced in vitality by the whole suppurative process.

Regarding the advisability of ligating the jugular in every case, that would bear a good deal of discussion. Some operators are very radical in the matter, believing that all cases should be ligated before opening the lateral sinus, to prevent the forcing of thrombi into the general circulation during the necessary manipulation of the sinus. Dr. Pierce does not agree with these men, but believes, however, that there is a distinct indication for ligating the jugular—not only ligating the jugular, but dissecting out a given piece of the jugular between the upper and lower ligatures. In this way we can to the best of our ability cut off the original source of the infection, because a thrombus occurs primarily from an infection of the blood vessel walls themselves, and the infection spreads beyond the thrombus in the vessel walls. He was reminded of a recent fatal case of thrombosis of the sigmoid sinus, where a piece of vein was removed well down to the calvicle. There was no thrombus here. The thrombus was away up just above the facial, and ceased there apparently, and yet there were microorganisms in the blood vessel wall itself.

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*See page 985.

Dr. Pierce could not see how the condition of endothelium of the intima of the inner wall of the sinus has very much practical bearing on these cases. Whenever a sinus is infected at all, this is pretty well destroyed. Its usefulness as a protective agent against the spread of the thrombus is at least rendered inactive. That is the main point upon which Dr. Holinger bases his thesis; that in order to prevent a thrombus from forming we should not disturb by our manipulative procedures the inner wall of the sigmoid sinus. When that has become thrombosed this point loses its value, it seems to Dr. Pierce.

The speaker's mode of procedure is in cases of sigmoid thrombosis to get free bleeding from both the upper and lower portion of the sigmoid sinus, if possible. If he fails in the lower portion, he does not necessarily proceed immediately, at the same operation, to ligate the jugular, unless the picture has been one of very grave pyemia before. In that case he would immediately proceed to the ligation and resection of the jugular, but after a day or so, if he finds that the sinus is still leaking pus into the general circulation, which is shown by the septic curve of the temperature, the occurrence of septic emboli, and general condition of the patient, then the jugular is ligated. However, at the time of the first operation, if the leucocyte count is high, and there is marked sepsis, the general condition of the patient impressing one with the fact that the patient is very ill, or septic infarcts are occurring, then he would immediately proceed to ligation of the jugular. If the case is not of this type, however, he might wait a day longer.

The best we can do in these cases is to establish in that sinus a nonseptic thrombus, by whatever means of operating we adopt, and it may be that if we have removed the greater portion of the offense, even if we do not get bleeding from below, there may be established a nonseptic thrombus at the lower end in the direction of the jugular bulb. But there is no doubt, from his experience, that if this does not prove to be a nonseptic thrombus at the lower end, the advisability of tying the jugular is proven by practical experience.

Dr. Pierce would like to know how many cases of septic thrombosis of the sigmoid or lateral sinus Dr. Holinger has seen and treated by the method described, and what his percentage of recovery is.

Dr. George W. Boot, since the first of last December, has operated on fifty-one mastoids, six of which had sinus thrombosis. Three of the sinus thrombosis cases died, all because they were not operated in time. One was a case of a type which the speaker imagined Dr. Holinger referred to. The boy had a metastasis in the great toe and a septic endocarditis. He died on the operating table. The other two cases had meningitis, as postmortem examination showed.

Dr. Boot believes that in these cases of sinus thrombosis, where you have a septic thrombus, as shown by the temperature curve, the sinus should not only be opened, but the jugular should be ligated. To do otherwise is to lock your stable door after the horse is stolen. The object of operation is to prevent the entrance of further infection into the system. These cases almost invariably have blood clots in the sinus, and in the majority of cases the organism causing the sinus thrombosis is the streptococcus. It is not a good idea to have too many streptococci loose in the system. They may cause trouble. The greater the number you keep from getting into the circulation, the more good you are doing the patient. He believes that in all these cases where there is a septic thrombosis the vein should be ligated. He was reminded of a case of a young man who had a mastoid operation; later he developed symptoms of sinus thrombosis; the sinus was opened and the pus evacuated; bleeding was secured from the upper and not from the lower end. Symptoms of septicemia continued. Finally, the speaker exposed the jugular to ligate it. He found it full of pus—three drams being evacuated from the jugular vein. The pus had extended so far down that he could not get below to ligate, and had to content himself with simply draining the jugular vein. The patient recovered after having a large abscess over the left trochanter major.

Dr. Boot passed around a number of temperature charts of sinus thrombosis cases. One case ran from early in January until perhaps about the end of April before being discharged finally. In another case, where the sinus was opened, drained and the jugular vein not ligated, the symptoms continued for several days after the first operation. Then the jugular was exposed and found filled with an organized thrombus. Another case was a woman who had been operated on by an otologist in New York City, who did a mastoid operation on her. She came back to Evanston with the wound not quite healed and came to the speaker for dressings. He dressed it. The next day she had a sharp chill, with a temperature of 103.6° F. There were no other symptoms evident. It looked as though she were developing a sinus thrombosis. It turned out to be a case of erysipelas. Another case illustrated the course of mastoid operation in scarlet fever. The chart showed very irregular temperature.

Dr. H. Kahn asked Dr. Holinger if he had understood him to say that the swelling of the eyelid was a symptom of late sinus thrombosis.

About a year ago there came to the speaker's notice and into his service a woman with a neglected mastoid. She was operated, and the sinus accidentally wounded. He packed it with gauze in the orthodox way. In the course of a week, one evening his attention was called to the swelling of the face, and a swelling of the eyelid, on the side of the mastoid operation. In the course of an hour or so a chill occurred, which recurred the next day. The wound was reopened, and the sinus was found thrombosed. Bleeding was secured from both sides, the wound packed, and she was treated in the usual method, and the patient recovered. This is in direct contradiction to the fact, stated by Dr. Holinger, that swelling or edema of the eyelid and swelling of the face is a symptom of late thrombosis. The speaker saw this symptom develop under his eyes.

Dr. Joseph C. Beck wished to speak from the few years' experience he had had with these cases. First, as to his observation when abroad, training under Zaufal and Piffl, who argued very much in favor of ligation, as most men are doing at the present day. On the other hand, Kaufmann, with a larger material, follows the method mentioned by Dr. Holinger, a method followed by many abroad even to this day, and he claims to have the best results. He could remember eight cases distinctly, five of Kaufmann's, on the Bohemian side, and three of Zaufal's, on the German side of the clinic, that were not ligated and recovered.

When Dr. Beck first came back he operated in a conservative way on these sinus cases, with not a very good result. But latterly he has been ligating, opening the sinus, and packing off the torcular region, and going down towards the bulb, being insistent on getting the bleeding from below, as near the jugular bulb as he possibly can. Then, if he does not get bleeding, he ligates the jugular, not necessarily resecting, unless there are distinct evidences of a thrombosed vein. As a means of prevention, he can see nothing better than that. Ligation is very easily done, and it is a safe procedure, which the speaker practices with excellent results.

As to the remarks regarding swelling of the face, we see that very frequently from the cutting of the temporal vein following ordinary mastoid operations.

Dr. Kahn's remarks reminded him of two cases he had had during the last six months, in which he had accidentally wounded the sinus. He was in great trepidation that something was going to happen, because he did not do anything to the sinus except cover it up. However, nothing happened in one case. In the other, however, there was a general multiple arthritis with symptoms of a septic process, but the patient recovered without any impairment to the joints. He did not know whether that was purely a toxic condition from the wounding of the sinus, or the sepsis already existing in the blood. He did not make any blood cultures previous to operation in this case.

Dr. Holinger, closing the discussion, said it was entirely sufficient for him that he had put an idea in the minds of the members, and he knew where they would land.

As to Dr. Kahn's question, Dr. Beck had answered it already. Swelling of the face and swelling of the eyelids are not identical. In the late course of the disease the swelling of the eyelid indicates a blocking of the sinuses on the base of the brain and of the veins which drain the blood from the orbit and eyelid. It is a different proposition from a swelling of the face.

Dr. Beck had said that he saw Professor Zaufal practice the method described in the paper. That is true. Dr. Holinger has operated for more than ten years according to it, but the experimental proof dates only since the last few years. He gave the abstracts of the papers which contained reports of the fundamental experiments. They contain points which you cannot evade. If experiments show that as soon as we remove the septic material from around a sinus the sinus is able, if not otherwise injured, to take care of the septic material within it, what more do you want? The indications which we must follow are: Remove all the septic material from around the sinus, and otherwise leave the sinus alone. Do not handle it. Do not puncture it. Do not work any more than is absolutely necessary, because of the danger of secondary thrombophlebitis. According to postmortems, this is one of the occurrences that has killed many patients after otherwise successful operations for sinus thrombosis. Experiments have shown that new thrombi in sinuses where septic thrombi have been before are, of course, infected. New thrombi, therefore, have to be avoided under all circumstances. It is not a step backward to limit and simplify an operation. The method described is easier, simpler and much safer.

One more important point must not be forgotten, namely, we have now better means of diagnosis, by leaving out all other points and relying alone on temperature and septic attacks.

We formerly had many of the late cases to deal with. The late, chronic cases have to be handled differently from those developing in connection with acute suppurations of the middle ear, where we are in a position to make a comparatively early diagnosis, and therefore can operate much earlier. Dr. Holinger does not, under all circumstances, object to the ligation of the jugular vein, but he does not think it is indicated in sinus thrombosis occurring in fresh acute suppurations of the middle ear. It is the latter which he is considering, and not the cases developing in connection with old cholesteatoma. He came to the conclusion that regular temperatures must be taken in every case of acute suppuration of the middle ear. In this way he discovered several acute cases during the last spring. After operation there are again rises of temperature, but at no time is there any question whether or not the patient is going to recover.

As the speaker had said in his paper, he did not expect the members to take his word from the start, but he wished to impress on them the fact that it is worth while to follow up the literature on the question. The operation is infinitely simpler and quicker, and if you have ninety-two per cent of recoveries, as Scheibe has with this operation, that seems a pretty good record.

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